THE USE OF
A RIGHT PECTORAL LEAD ELECTROCARDIOGRAM
IN MYOCARDIAL INFARCTION

A THESIS

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C O N T E N T S

INTRODUCTION

At the beginning of the present century, in 1903, Einthoven published his account of the string galvanometer. It was not, however, for about a decade that such instruments came into general hospital use.

The syndrome of coronary artery occlusion was first described by Herrick in 1912. The condition had long been known to pathologists and was commonly regarded as a cause of sudden death, but it had not been realised that it was compatible with survival. A further decade elapsed before recognition of the syndrome became common (MacNee, 1925; Hamman, 1926; Parkinson & Bedford, 1928b; Levine, 1929).

It may thus be seen that the clinical recognition of coronary artery occlusion and the introduction of the electrocardiograph as a diagnostic aid in medicine occurred more or less simultaneously.

The electrocardiographic changes accompanying coronary artery occlusion were investigated in 1918 by

Smith, at Herrick's suggestion. He used the three standard limb leads and described T wave changes in dogs following coronary artery ligature. A year later, Herrick reported a case of coronary thrombosis in a patient whose electrocardiogram resembled those of Smith's dogs (Herrick, 1919). Smith described serial changes in the electrocardiogram in further experimental work (Smith, 1920; 1923).

Earlier than this, Eppinger & Rothberger (1909) had shown that the injection of silver nitrate solution into the posterior wall of a dog's heart produced ST elevation in leads II and III and ST depression in lead I. Pardee (1920) reported a case of coronary thrombosis with electrocardiographic changes similar to those of Eppinger & Rothberger, and postulated that the resulting infarct involved the posterior wall of the heart. These ST deviations came to be known as Pardee's sign (and should therefore only be so termed when they show the pattern of posterior infarction).

Serial changes in the human electrocardiogram following coronary artery occlusion were described by Parkinson & Bedford (1928a). They showed that those leads displaying ST elevation at an early stage, later showed an iso-electric ST segment with an inverted T wave.

Conversely ST depression gave place to an iso-electric ST with a tall upright T. On this basis, they were able to distinguish two distinct electrocardiographic patterns following coronary artery occlusion: the T_1 in which T_1 was inverted and T_3 upright; and the T_3 in which T_1 was upright and T_2 and T_3 inverted. This description of two types cleared up some confusion as to what changes occurred in the electrocardiogram. That these two types indicated different locations of the infarct was shown by Barnes & Whitten (1929) and experimentally in cats by Crawford, Roberts, Abramson & Caldwell (1932). The historical aspect is ably covered by Barnes (1940).

It would be advisable at this stage to comment upon the terms 'coronary thrombosis' and 'myocardial infarction'.

When the earlier work was carried out the two terms were regarded as synonymous. The title of Parkinson & Bedford's paper - "Successive changes in the electrocardiogram after cardiac infarction (coronary thrombosis)" - serves to illustrate this. It had been observed that anterior infarcts usually resulted from blockage of the anterior descending branch of the left coronary artery; that posterior infarcts resulted from blockage of the

right coronary artery. Hence there was a tendency to interpret the electrocardiograms of infarction in terms of the occluded artery. Whitten made a detailed study of the arterial supply of the myocardium by the celloidin - corrosion method (Whitten, 1928; 1930) and showed that the electrocardiogram indicated the site of the infarct, and that this was not invariably due to blockage at any particular place. Variations in blood supply to the myocardium were recorded and it was shown that posterior infarcts, for instance, might result from blockage of the circumflex branch of the left coronary artery.

Saphir, Priest, Hamburger & Katz (1935) showed conclusively by careful dissection of the coronary arterial tree that where "a myocardial infarct was encountered, at least two branches of the coronary arteries supplying the infarcted area were involved." The inference was that a single coronary occlusion could be compensated by anastomoses and would be symptomless as there would be no muscle necrosis. It is thus appreciated now that the terms coronary thrombosis and myocardial infarction are not synonymous. In a recent detailed investigation of the correlation between ante-mortem electrocardiographic findings and post-mortem findings, Myers, Klein & Stofer (1948) state, in their preamble: "... a description of

the pathologic findings pertaining to the coronary vessels is also omitted since the electrocardiographic abnormalities are a direct manifestation of the secondary changes in the myocardium rather than the primary changes in the coronary arteries".

To return to the account of the development of electrocardiography in relation to myocardial infarction, it must next be recorded that cases were observed where undoubted myocardial infarcts failed to show convincing electrocardiographic evidence.

Wolferth & Wood (1932) realising that the conventional leads lay in the frontal plane of the body decided to explore the sagittal plane. This they did by transferring the right arm electrode to the precordium (in the fourth left intercostal space) and the left arm electrode to the back just below the angle of the left scapula. The left leg electrode remained in that site. By using these connections and turning the lead selector switch of the electrocardiograph to leads I, II and III, they recorded what they termed leads IV, V and VI respectively. As a result they were able to show that such electrocardiograms were a valuable adjunct to the conventional leads and especially in the diagnosis of myocardial infarction where the limb leads were normal or equivocal.

They followed up this work by showing that it was possible to produce myocardial infarcts in dogs by coronary artery ligature without producing significant changes in the limb leads. Precordial leads, however, supplied the required information (Wood & Wolferth, 1933).

Liberson & Liberson (1933) used the Wolferth lead IV with reversed polarity (left arm electrode on precordium; right arm electrode on back) for, by doing so, the electrocardiogram resembled in its general contours those of the standard limb leads. Katz & Kissin (1933) made a study of lead IV and stated: "... we decided to follow the lead IV already studied by Wolferth & Wood. ... in following their procedure we were tempted to reverse the connections of the electrodes because the normal configuration of lead IV so taken is like that of the other leads."

This, then, was a time when hardly any two sets of workers used the same technique. Roth (1935) gave an account of the use of chest leads up to that date and made a critical review of them. He pointed out that Wolferth's leads IV and V were invariably similar and produced evidence that the site of the distant electrode is relatively unimportant. Lead VI (scapular region - left leg) was valueless and being given up. Roth

emphasised the importance of accurate positioning of the precordial electrode as small errors could produce large changes in the electrocardiogram, and in fact recommended the use of three chest positions - right pectoral, left pectoral and apical. These chest leads have been used by Wood & Selzer (1939).

The importance of accurate positioning of the precordial electrode had previously been emphasised by Wilson (1930) and by Hoffman & Delong (1933). The site of the apex thrust was regarded as the optimum position by Edwards & van der Veer (1938) but in cardiac hypertrophy they thought that the electrode should never be taken further to the left than the anterior axillary line. Robinson, Contratto & Levine (1939) investigated the optimum site for the precordial electrode and concurred that the apex was the most useful point.

It was clear that some sort of standardisation of technique was necessary; and in 1938 the report of a joint committee representing the American Heart Association and the Cardiac Society of Great Britain and Ireland was published. This recommended the use of an apical electrode paired with either a right arm or left leg distant electrode. The polarity was to be so arranged that relative positivity of the precordial electrode

resulted in an upward deflection in the electrocardiogram. Wolferth's original lead IV was thus reversed. The American Heart Association made a supplementary report (1938) standardising six positions for precordial leads when more than a single (apical) lead was required.

Wilson and his colleagues have approached the subject of precordial leads from a different standpoint. Leads taken from the surface of the heart have been shown to be similar to those taken from the overlying positions on the precordium (Wilson, 1930). This fact has been utilised in the investigation of bundle branch block and myocardial infarction. The precordial leads were recorded from an oblique line perpendicular to the surface marking of the interventricular septum (Wilson, MacLeod, Barker, 1932; Wilson et al. 1944; Wilson, Rosenbaum & Johnston, 1947). In 1934 and 1935 there appeared a series of four papers on the form of the precordial electrocardiogram in experimental myocardial infarction in dogs (Wilson, Hill & Johnston, 1934; Johnston, Hill & Wilson, 1935; Wilson, Hill & Johnston, 1935; Wilson, Johnston & Hill, 1935). In this work the distant electrode was placed on a hind leg. Shortly before these papers appeared Wilson, Johnston, MacLeod & Barker (1934) published an account of a new distant electrode.

Lead wires from the three limbs were combined in a central terminal which theoretically remained at zero potential throughout the cardiac cycle and which in practice only showed very small potential variations. The electrocardiogram was recorded by leading from the precordial electrode (or indeed from a limb) to the central terminal and displayed only the potential variations of the former. Such electrocardiograms were called 'unipolar'; and unipolar electrocardiography was developed by Wilson and his colleagues during the ensuing years, gradually being adopted by more workers until, at the present, such a technique is in almost general use. A comprehensive account of the use of unipolar limb and precordial leads in determining heart position, predominant ventricular hypertrophy, bundle block and myocardial infarction appeared in 1944. Wilson had served on the committees mentioned above and accepted their recommendations.

The Object of the Present Work

Precordial leads reveal changes in the underlying muscle (Wilson et al. 1944). Hence they are of particular value in the diagnosis of anteriorly placed infarcts. In the case of posterior infarcts, the precordial leads may show changes but do not do so usually. The ideal lead is one placed in the oesophagus, but this is not very acceptable to patients. Dorsal leads are distant from the epicardial surface and cannot be expected therefore to show variations comparable with the precordial leads. That they hardly figure in the literature lends force to this supposition. Curiosity as to the configuration of the dorsal leads in normal and infarcted hearts and, in particular, the possibility that dorsal leads might show evidence of posterior infarcts led to the following investigation.

Method

The six precordial lead positions originally recommended by the American Heart Association were chosen, and four further lead points were added in the following manner:

the horizontal line drawn from point 4 through point 5 to point 6 was continued round the thorax and it intersected -

the posterior axillary line at point 7

the left posterior scapular line at point 8

the midline at point 9

the right posterior scapular line at point 10.

It may be briefly stated that the results obtained were disappointing; and it was decided to continue the number of lead points to encircle the chest. The ten lead points enumerated above lie roughly on a plane, mainly horizontal, but obliquely placed in the thorax. It was decided to retain the subsequent lead points in the same plane.

Point 12 was chosen high in the right axilla

Point 11 lay midway between 10 and 12

Point 13 lay midway between 12 and 1.

These points are illustrated in the accompanying figures.

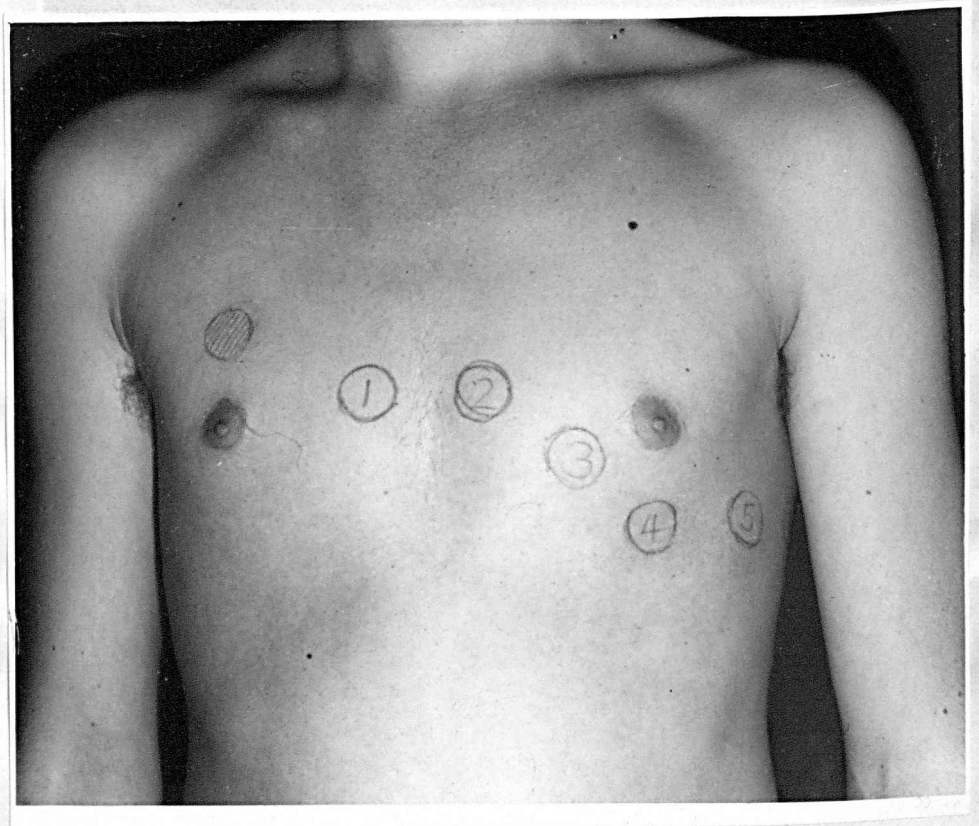


Figure 1. The precordial lead positions. Position 13 is shaded; 12 is not seen.

It may be seen from Figure 3 that point 11 lies on the muscle mass forming the posterior fold of the right axilla. Point 13 lies at the intersection of the right mid-clavicular line and the third right intercostal space. Points 8 and 10 each lie about one inch below the inferior angles of the left and right scapulae.

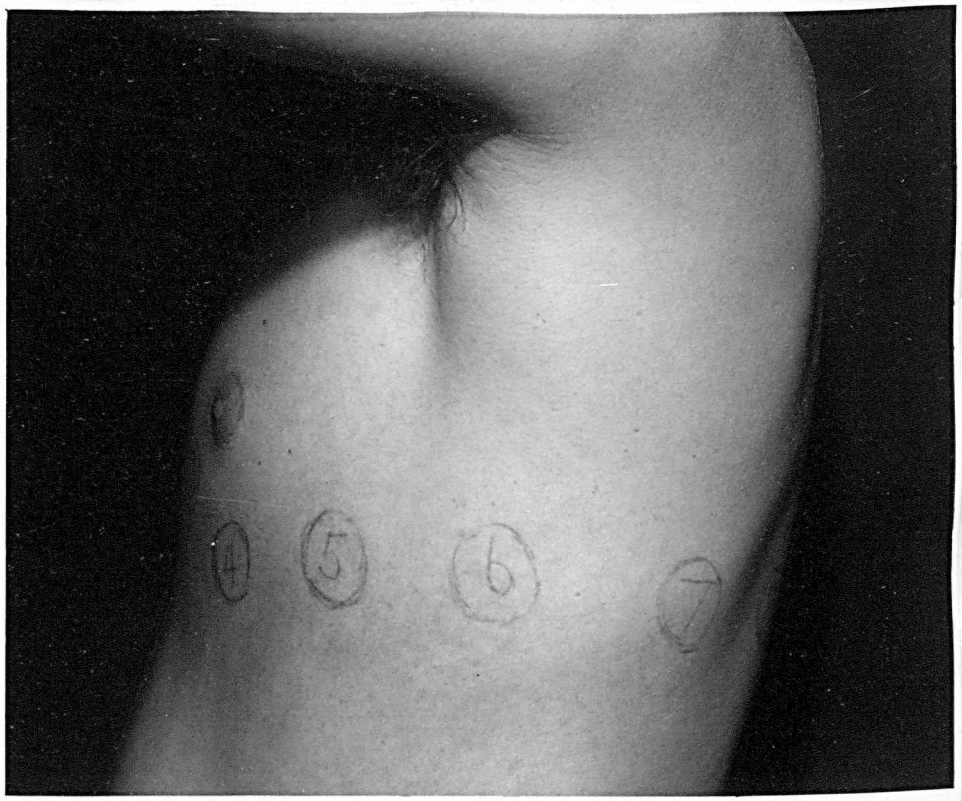


Figure 2. Left lateral aspect.

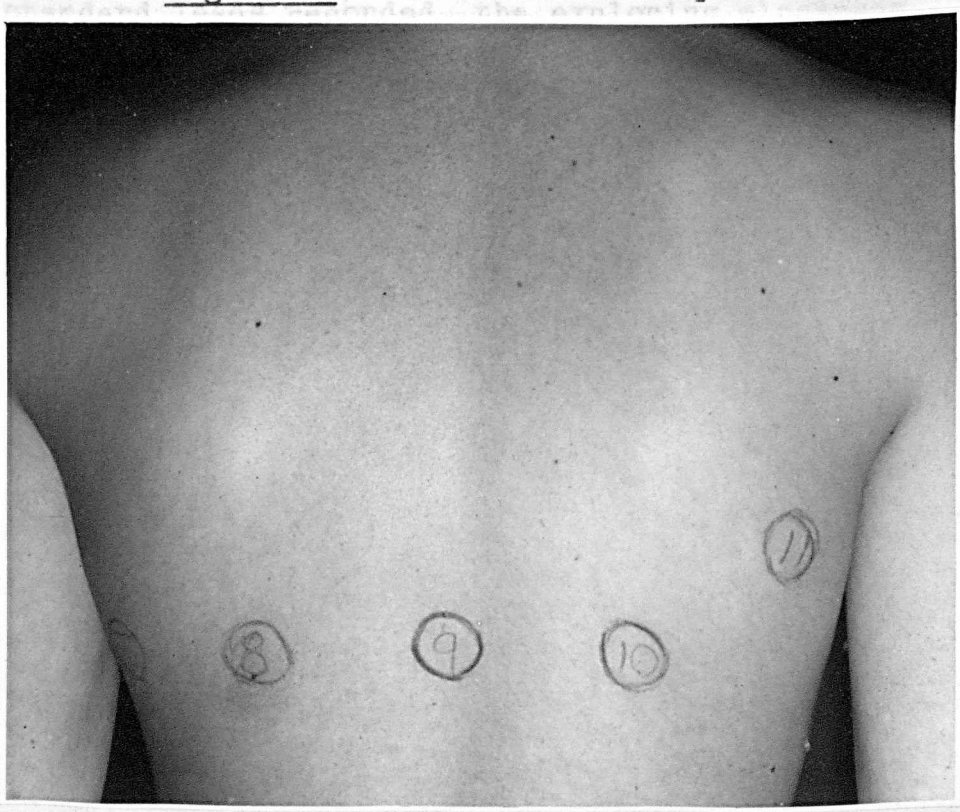


Figure 3. Posterior aspect.

The indifferent electrode was placed on the left leg. These chest leads therefore belong to the CF series and have been designated CF 1 to 13 consecutively.

The electrocardiograms were taken in the sitting position.

The electrodes were made of tin-plated brass, were circular in shape and measured 3 cm. in diameter. They were made to the specifications of Marchant and Wallace-Jones (1940).

The chest positions were marked in each patient with a skin pencil before the records were made. After having standard leads recorded, the exploring electrode was placed on each of the chest positions in turn, the electrode being either held in place by hand (rubber gloved) or held in an insulated holder. The skin was prepared with Cambridge electrode jelly. The galvanometer was a Cambridge string instrument, and was standardised for normal sensitivity (1 millivolt = 1 cm. deflection) before each lead was recorded.

It was not always easy to obtain a level base line. This was noted particularly in the left axilla (positions 5, 6 and 7). Respiratory movement of the ribs caused some movement of the electrode with varying

contact, and this is the probable explanation. When this occurred, the skin surface and electrode were cleaned, jelly was re-applied and the patient instructed to breathe more gently.

RESULTS

The subjects investigated by leading from the thirteen positions in the manner described above, comprised:-

A. 23 normal individuals (mostly medical students).

B. 19 cases judged by the clinical features and by the standard lead electrocardiograms to have sustained posteriorly placed myocardial infarcts. In this group a total of 33 circuits of the chest were made.

C. 13 cases judged by similar criteria to have sustained anterior infarcts (20 chest circuits).

D. 14 miscellaneous cases with 17 circuits. These had standard lead electrocardiograms showing various types of T wave abnormalities; but none were thought to have had a myocardial infarction.

Group A.

This group consisted of 20 male medical students aged 20 to 24, and three hospital patients with peptic ulceration aged 20, 23 and 26. All had normal hearts on clinical examination; and ten cases were found to have normal cardiac contours by x-ray screening.

The individual deflections in each of the thirteen leads were measured and the results are shown in Table I.

The set of electrocardiograms from the subject whose record was most nearly average is shown in Figure 4.

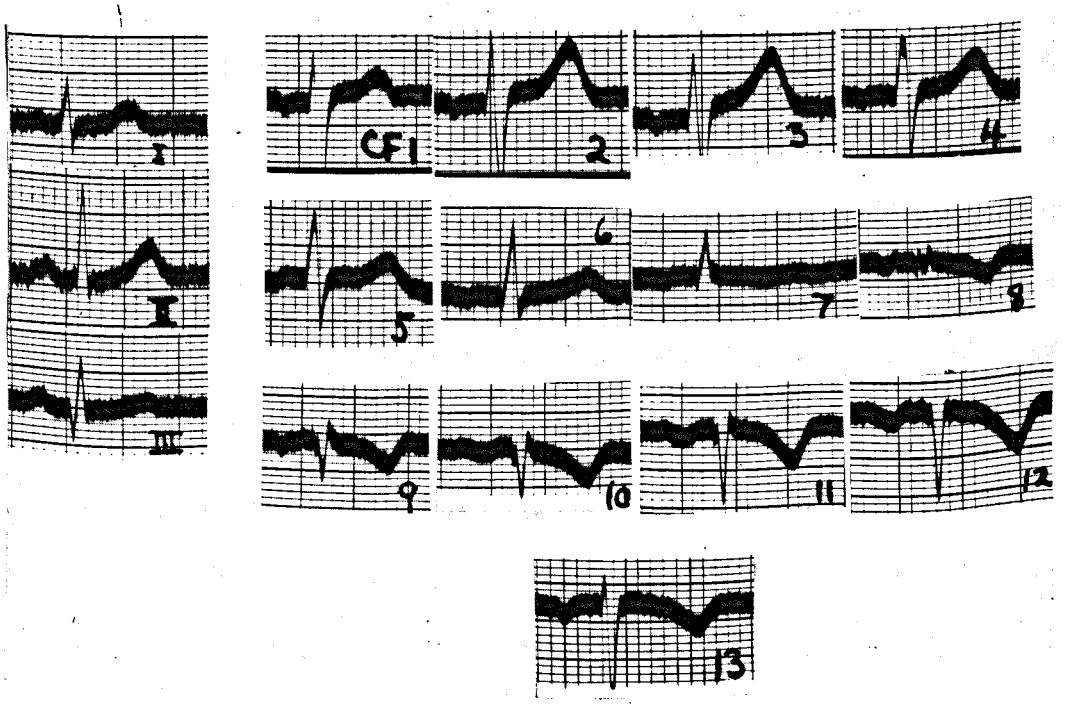


Figure 4. Normal leads

TABLE I

	Q			R			S			T		
	Av.	Max.	Min.	Av.	Max.	Min.	Av.	Max.	Min.	Av.	Max.	Min.
CF1	0	0	0	5	10	1	-13	-17	-9	3	8½	-4
2	0	0	0	7	12	2	?	?	-8	7½	11	2
3	0	0	0	8	15	2	?	?	-5	8	13	4
4	0	-1	0	8	15	2	-6	?	-2	6½	13	3
5	0	-2	0	8	17	3	-5	-12	-1½	4	10	2
6	-1/3	-2	0	7	14	3	-3	-9	0	3	6	1
7	-1½	-6	0	3	7	0	-½	-5	0	0	3	-3
8	-3	-8	0	3	8	0	-½	-7	0	-1	+2	-5
9	-5½	-10	0	2	6	0	0	-9	0	-3	½	-7
10	-6½	-11	0	1½	4	0	-½	-12	0	-3	-1½	-7
11	-5½	-12	0	1	3	0	-4	-14	0	-4	-1½	-9
12	-5½	-12	0	1	2	0	-5	-14	0	-3½	-1½	-8
13	0	0	0	2	5	1	-12	-15	-10	-3	-1½	-6

(Measurements in millimetres)

Notes:

1. The S wave in leads CF1, 2 and 3 was too deep to be measured (it frequently went off the record). The average figure of -13 mms. in CF1 is an approximate one only. The average figure of -6 mms. in CF_4 is the mean of 15 results. It should be larger, as the remaining eight figures were all greater than that.

2. In CF_9 the average R wave is shown as 2 mm. It was zero in 5 cases owing to the presence of a QS wave.

3. U waves occurred in 12 cases as follows:
   CF1 - 3 times. CF3 - 9 times.
   CF2 - 10 times. CF4 - 5 times.
   The maximum deflection was 1 mm. and was seen in leads CF1 and CF2.

Group B.

This group consists of records from 19 cases. In each the diagnosis of myocardial infarction had been made clinically. The records are similar in that:

a. the standard leads show a deep Q3 with an inverted T in leads II and III;

b. the precordial leads generally show a lerger upright T than normal.

A typical example is shown in Figure 5.

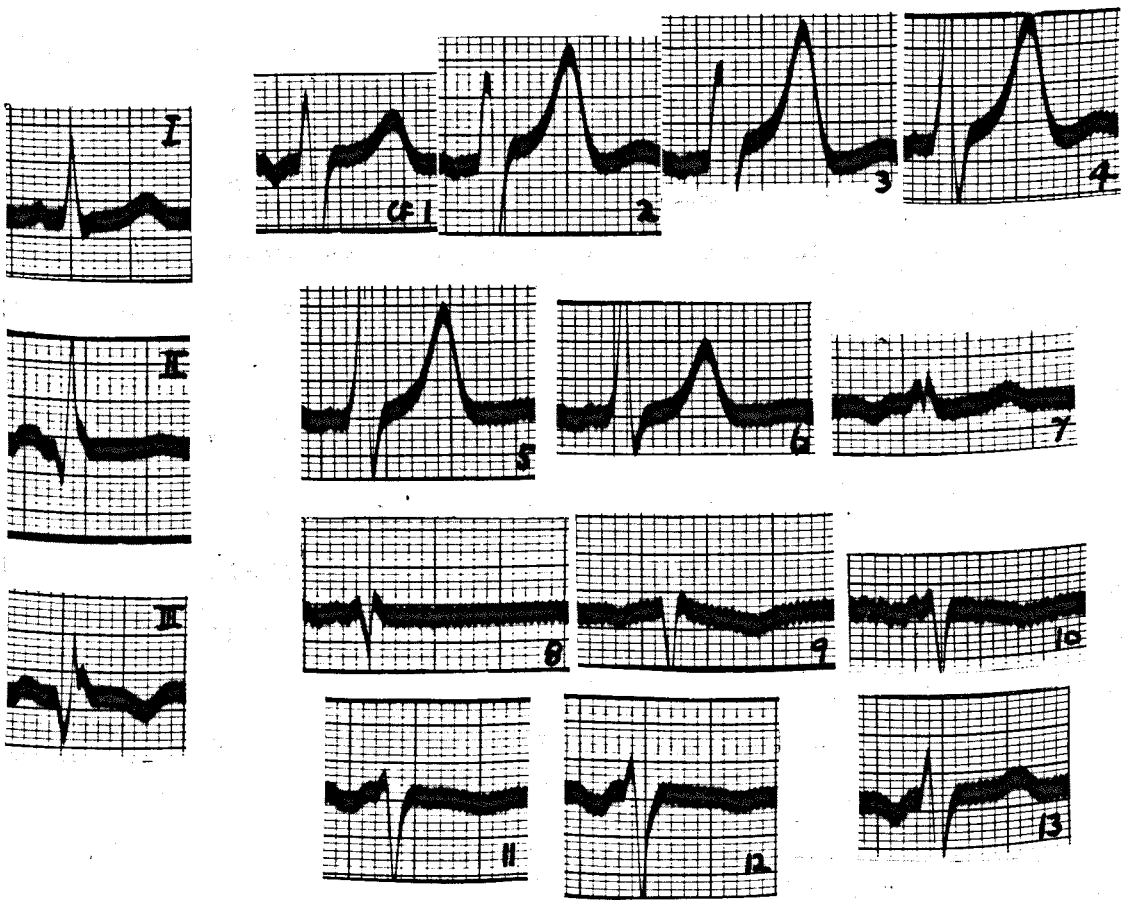


Figure 5. E.C.G. Record of a Posterior Infarct.

The components of the QRS complex have not been measured as they are subject to variable factors such as ventricular hypertrophy and the position of the heart. This latter factor also, of course, applies to Group A records. It was possible, however, after ST segment deviations had subsided to measure T wave amplitudes and these are shown graphically in Figure 10.

Group C.

This is a group of 13 cases of anteriorly placed infarcts, and like the previous group, came from patients in whom the diagnosis was a clinical one. These could be arbitrarily separated into three sub-groups as follows:-

a. 5 cases. The chest leads show maximal changes in the zone CF1 - CF3. Such records are generally regarded as examples of antero-septal infarctions and one is shown in Figure 6.

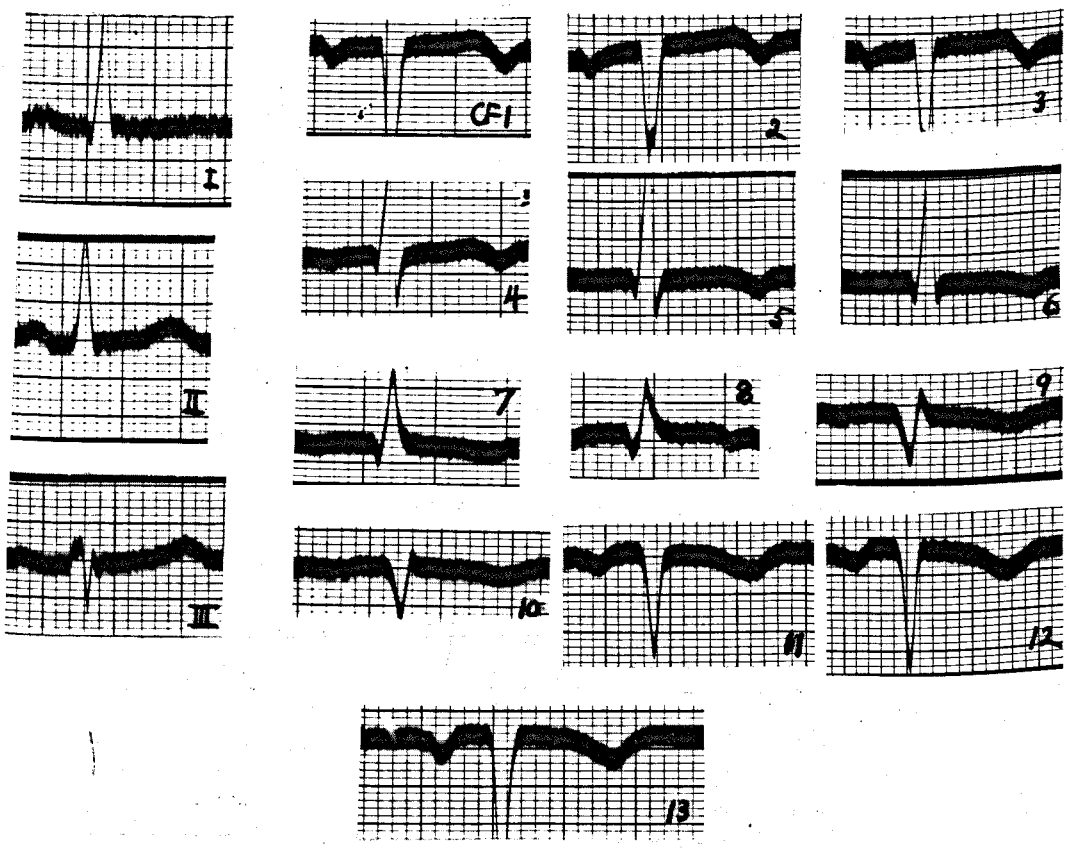


Figure 6. Antero-septal infarction.

b. 6 cases. The chest leads show maximal changes in the zone CF4 - CF6 and may be described as antero-lateral infarction. Figure 7 illustrates such a case.

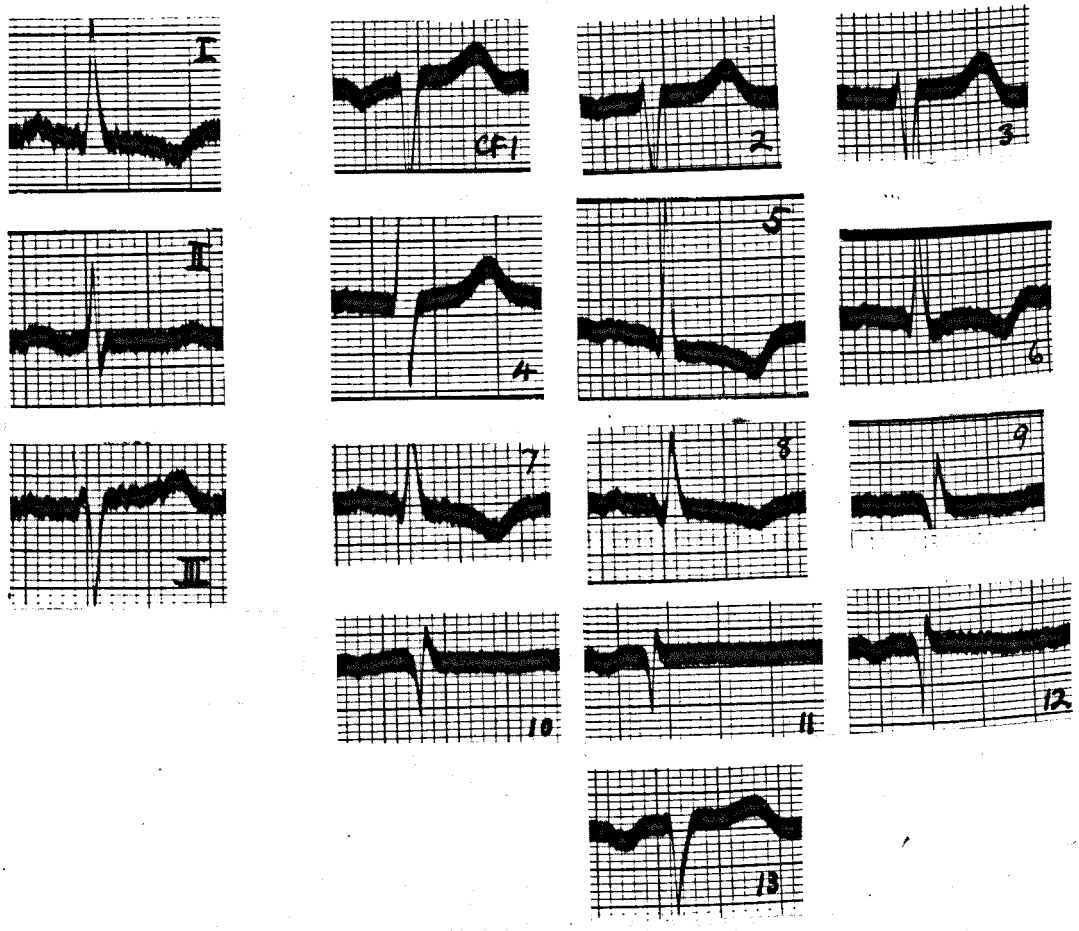


Figure 7. Antero-lateral infarction.

c. 2 cases. The standard leads showed T wave inversion in leads I and II in one case (Figure 8) and in all three leads in the other (Figure 9).

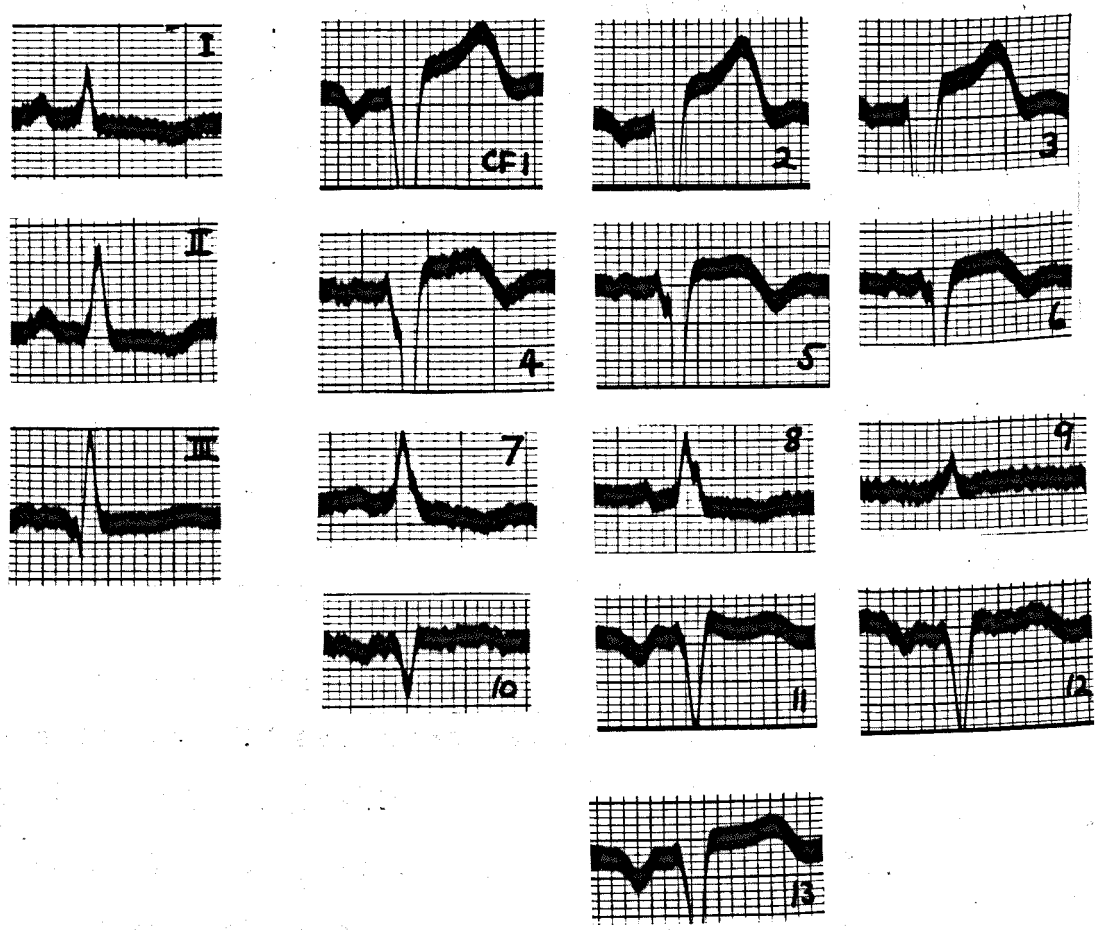


Figure 8. The chest lead patterns associated with negative T1 and T2.

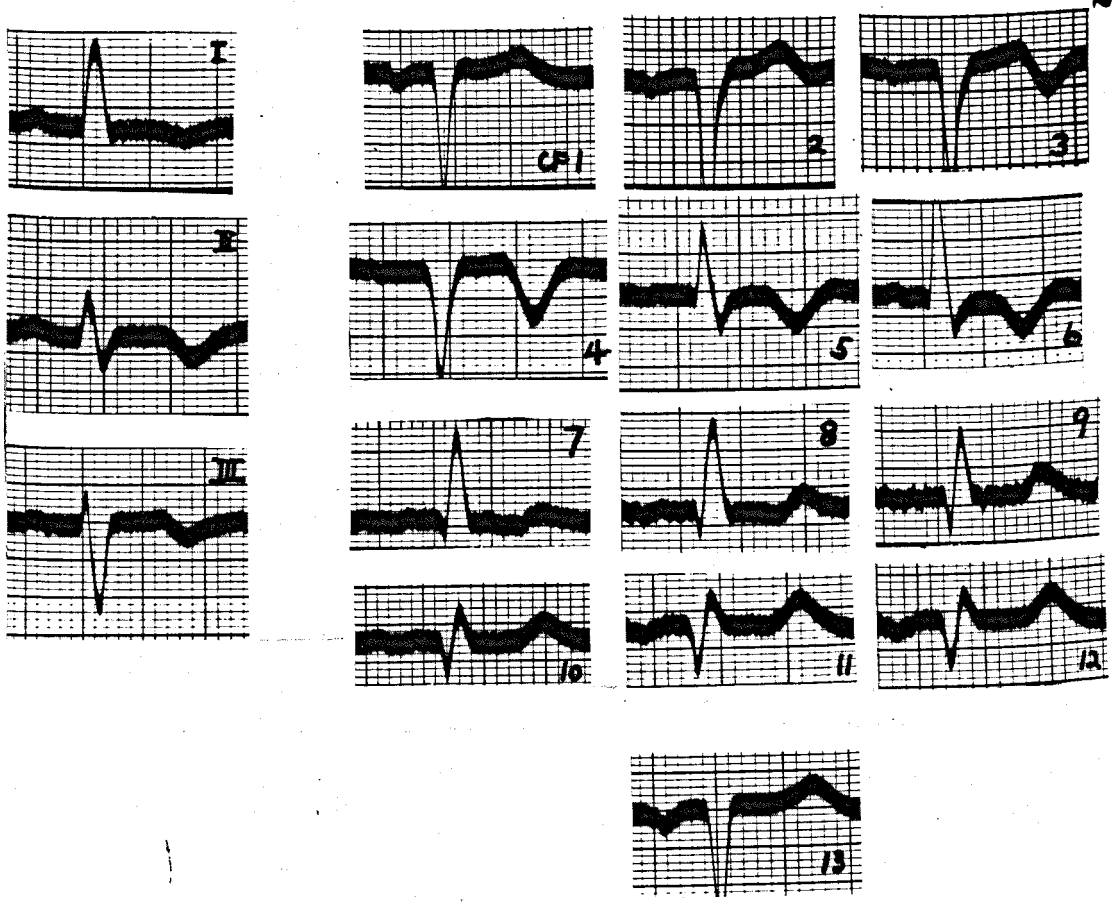


Figure 9. The chest lead patterns associated with negative T1, T2 and T3.

The average T wave amplitudes for each of leads CF1 to CF13 are shown separately for sub-groups (a) and (b) in Figure 10. In some cases the T wave measurements have been approximations owing to there being ST segment displacements. The T wave amplitudes from sub-group (c) have not been shown graphically because only two cases are included. It will suffice to draw attention to the fact that the T wave in CF13 is upright in both.

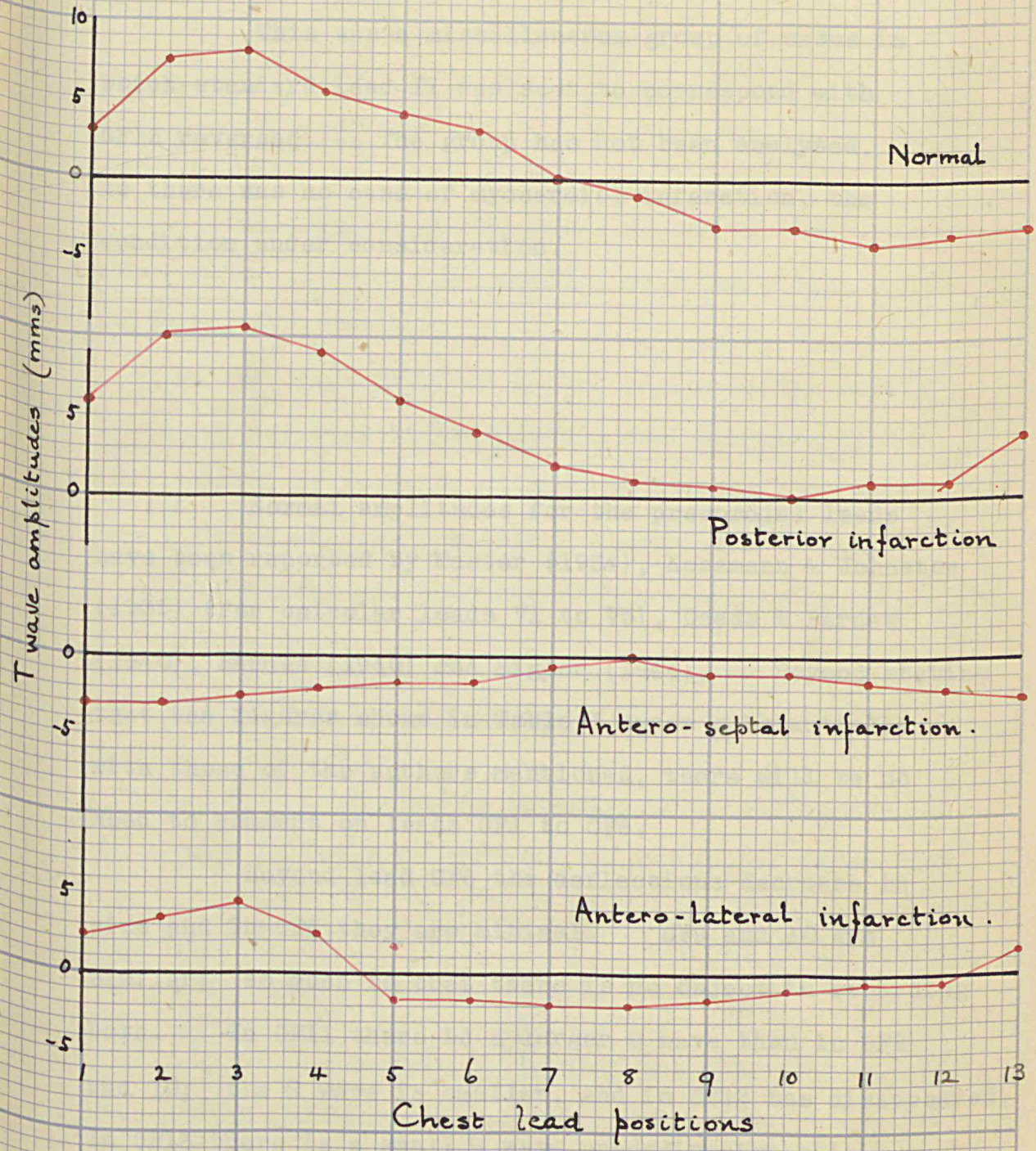


Figure 10. T wave amplitudes in normal and infarcted hearts.

Group D.

This was a miscellaneous group of cases in which standard lead II was seen to correspond with CF13 reversed. The group has not been analysed, as there was no case of myocardial infarction, the condition under consideration.

Normal amplitudes for the precordial leads have been reported by Master (1934), Kossmann & Johnston (1935) (for unipolar leads V1 to V5), Deeds & Barnes (1940), Barnes (1940) and Sigler (1944). Beyond stating that the figures shown in Table I agree substantially with those of the authors mentioned, there will be no need to comment on leads CF1 to CF6.

Beyond lead CF6 the deflections decrease in size and in CF8 they are minimal. CF9 and 10 are similar; so much so that there is no object in recording both. In CF11 there is a larger Q wave than in CF9 and 10; the R wave is smaller, and in CF12 has disappeared. In CF13 there is a small R wave preceding the main

deflection which is downward.

CF12 approximates to standard lead II reversed; and was expected to do so from the situation of the electrodes.

Inspection of the examples and of the T wave sizes shown in Figure 10, enable one to make the following observations:-

1. CF leads from the dorsal region at the level of the body of the ninth dorsal vertebra (the level of position 4) show amplitudes too small to be of use in clinical electrocardiography at normal galvanometer string sensitivity.

2. Lead CF13 shows amplitudes comparable with those of the precordial leads.

3. T in lead CF13 is inverted in normal hearts and in cases of apparent antero-septal infarction.

4. T in lead CF13 is upright in cases of posterior and apparent antero-lateral infarction.

It was decided to investigate lead CF13 further with particular reference to autopsy evidence of myocardial infarction.

Leads CF13 and CF13i.

At this stage, it was noted that the normal lead CF13 showing an inverted P wave, a QRS complex with the main deflection downward and an inverted T wave, in its general contours, resembled Wolferth's original lead IV but with this difference: relative positivity of the exploring electrode compared with the distant electrode recorded a downward deflection in Wolferth's lead IV: it records an upward deflection in CF13.

The joint committee of the American Heart Association and the Cardiac Society of Great Britain and Ireland (1938) recommended, inter alia, that the precordial lead connections should be made in such a way that relative positivity of the chest electrode should result in an upward deflection in the electrocardiogram. To conform with this, Wolferth thereafter reversed the polarity of his lead IV. It was referred to as IVB to indicate that the indifferent electrode was placed on the back (near the inferior angle of the left scapula). The particular advantage was that the new lead IV resembled the complexes of the standard limb leads, and simplified its interpretation.

For the same reason, it was decided to reverse the polarity of lead CF13. The resulting lead is hereafter referred to as CF13i (i for inverted). The practical advantage is that the lead shows complexes resembling those of the standard limb leads: but it must be remembered that relative positivity of the chest electrode compared with the left leg electrode results in a downward deflection. The characteristic features of CF13 in normal subjects have been included in Table I. By reversing this lead, the following data apply to lead CF13i.

1. A large upright P wave.
2. A Q wave averaging 2 mm. with extremes of 1 and 5 mms.
3. An R wave averaging 12 mms. (extremes of 10 and 15 mms.)
4. No S wave.
5. An upright T wave of 3 mms (extremes of 1.5 to 6 mms.)

These figures are derived from 23 normal male subjects, aged 20 to 26. There was substantial agreement between the figures for leads CF1 to CF6 in Table I and those reported figures quoted above. It is claimed therefore that these characteristics of lead CF13i are representative of normal hearts.

The Interpretation of Lead CF13i.

This lead may be considered in two ways:-

1. If the electrodes are placed for recording standard lead II, and the right arm electrode is then transferred to the right side of the chest, a lead will be obtained which may be expected to resemble lead II in its contours. In practice, this is borne out.

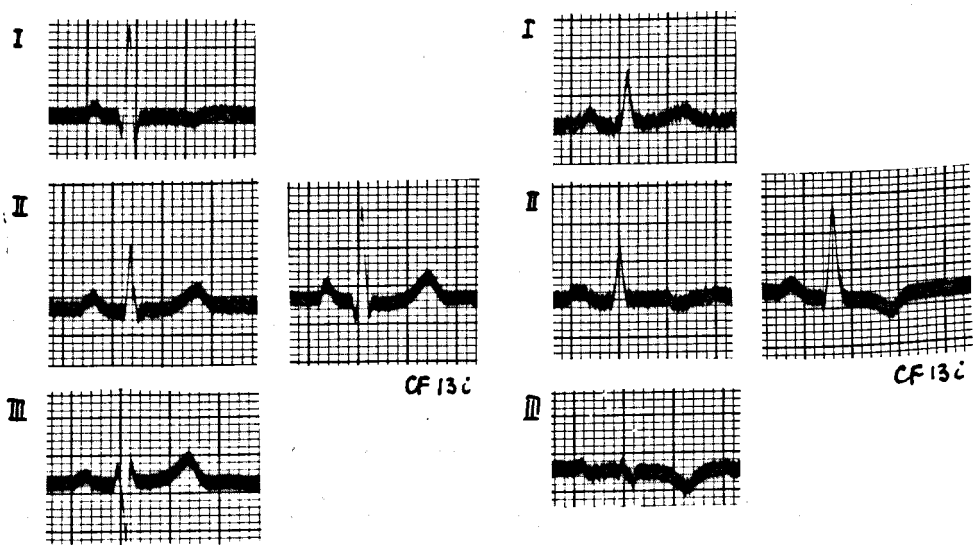


Figure 11. Two examples of CF13i with standard leads for comparison.

It does not follow, however, that CF13i invariably resembles lead II as Figure 12 shows.

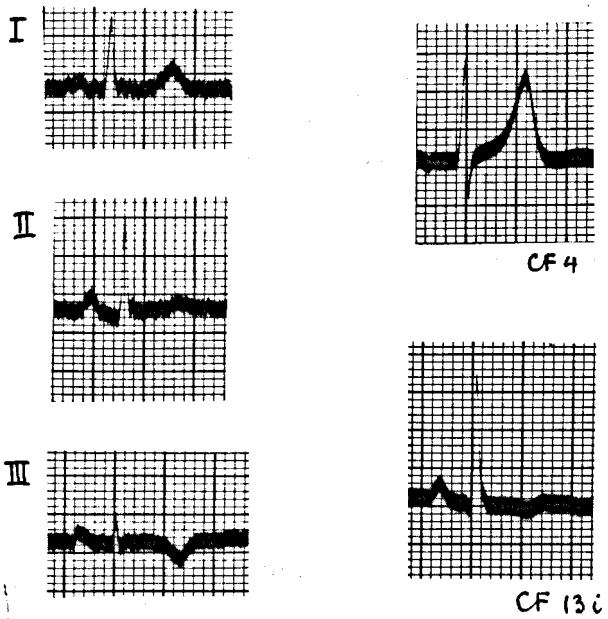


Figure 12.

Here T_2 is upright but T in CF13i is inverted. Such cases are unusual, but where T_2 is indefinite (e.g. diphasic or iso-electric) CF13i usually shows a T wave which may be interpreted clearly. Illustrations are supplied in Figure 13 (A, B, C, D and E) and all were obtained from clinical cases of myocardial infarction.

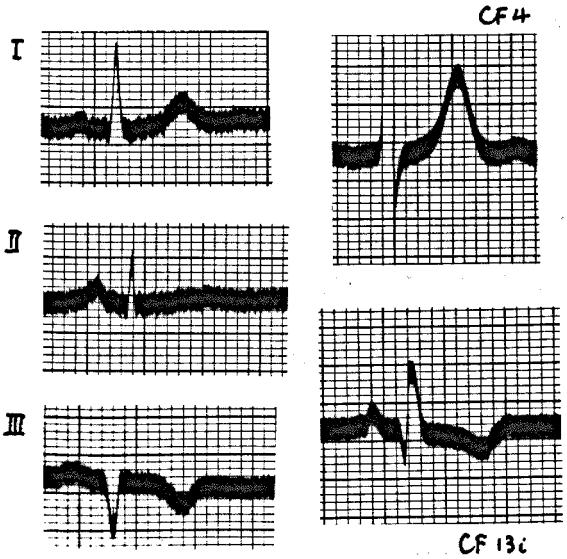


Figure 13 A.

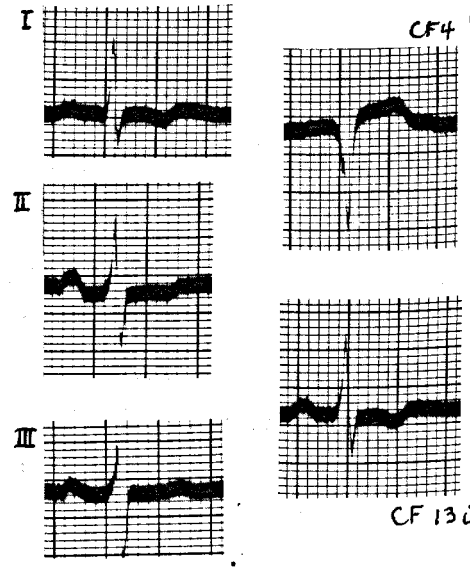


Figure 13 B.

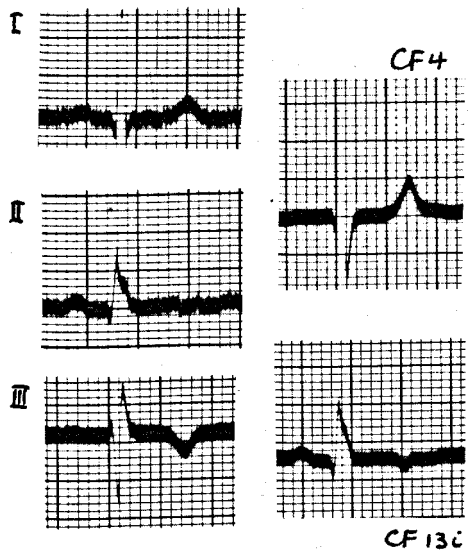


Figure 13 C.

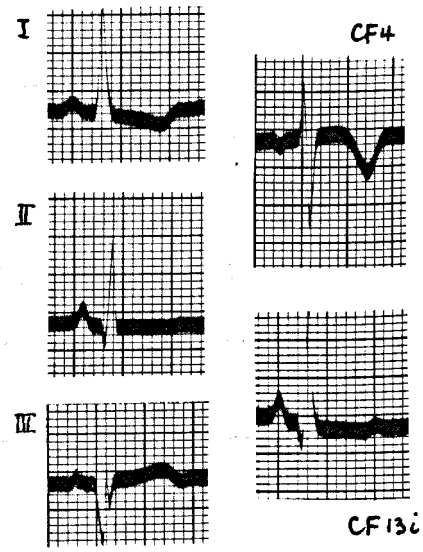


Figure 13 D.

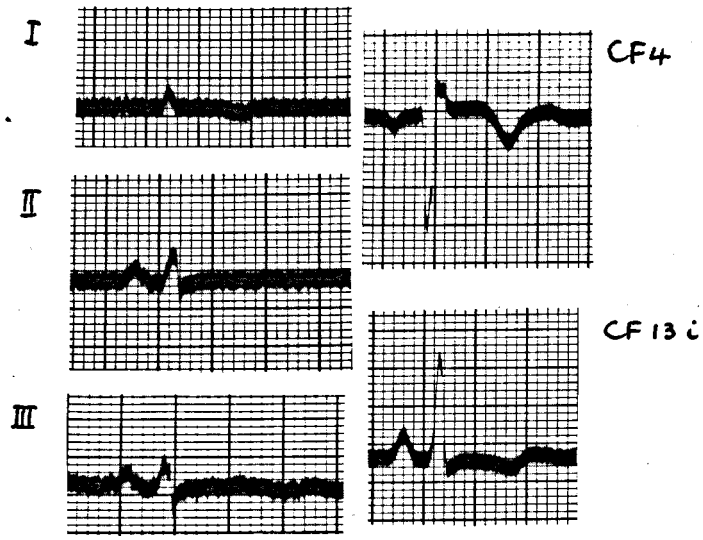


Figure 13 E.

The importance of T_2 in the diagnosis of posterior infarction has been emphasised by Evans & Hunter (1943) who stated that in 30 out of 32 cases investigated, a negative T_2 supplied the evidence necessary for the electrocardiographic diagnosis.

2. The alternative way of regarding CF13i is as follows:

Leads from diametrically opposite points on the thorax yield complexes which are the reverse of each other.

This is well illustrated by Bohning, Katz, Robinow & Gertz (1939) and Bohning, Katz & Langendorf (1941) who have encircled the chest using fifteen

different positions on each of two horizontal planes through the chest, in an investigation of ventricular hypertrophy and intra-ventricular block. Inspection of their illustrations shows that, as may be expected, leads from diametrically opposite points are the reverse of each other, and are further modified by the fact that those from the anterior chest wall show deflections of a greater magnitude than those over the posterior wall. Further evidence may be found in a paper by Rosenbaum, Hecht, Wilson & Johnston (1945) on the investigation of cases of the Wolff-Parkinson-White phenomenon; and by Goldberger (1944) who has studied axis deviation and ventricular hypertrophy using, in addition to the standard and unipolar limb leads, unipolar leads from 25 positions on the trunk.

Reference to Table I and Figure 4 also confirms that leads from diametrically opposed points are the reverse of each other. CF13 might therefore be expected to show complexes the reverse of CF7 for, as may be seen in Figure 14 these points are diametrically opposed. If CF13 be inverted by reversing the lead wires CF13i should resemble CF7. This latter lead, however, as Figure 14 also shows, is

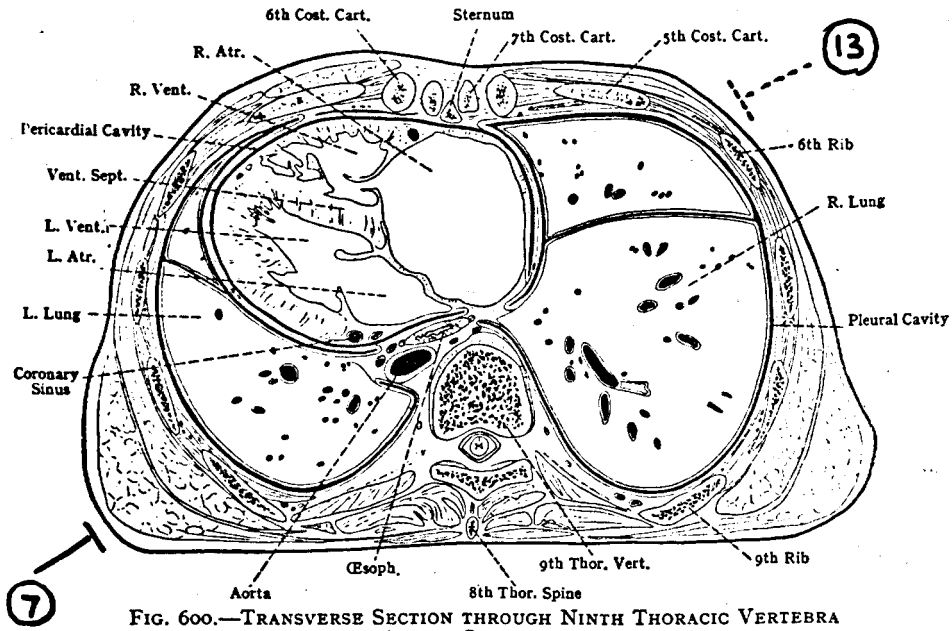


FIG. 600.—TRANSVERSE SECTION THROUGH NINTH THORACIC VERTEBRA (AFTER SYMINGTON).

Figure 14. Transverse section of the chest at the level of the body of the ninth dorsal vertebra. (Copied from Buchanan's "Manual of Anatomy", 8th ed. 1949).

distant from the heart, and shows smaller deflections, as indicated above (Bohning et al.), than, say, a lead from position 6.

Wolff (1950) referring to the precordial leads states: "Small R and deep S waves are seen in V1, and small Q, tall R and small or no S waves in V6." V6 and V7 are both left ventricular leads and in the latter a small Q, tall R and absent S

occurs even more frequently than in V6. It is noteworthy that the characteristics of V6 and V7 should also be shown by CF13i. Evans & Hunter (1943) have referred to the value of lead CR7 and a few records have been made in order to compare this lead with CF13i. Two examples are shown and these illustrate the evolution of these leads in cases of posterior infarction. The first shows the more striking changes in CF13i: in the second the two leads are similar.

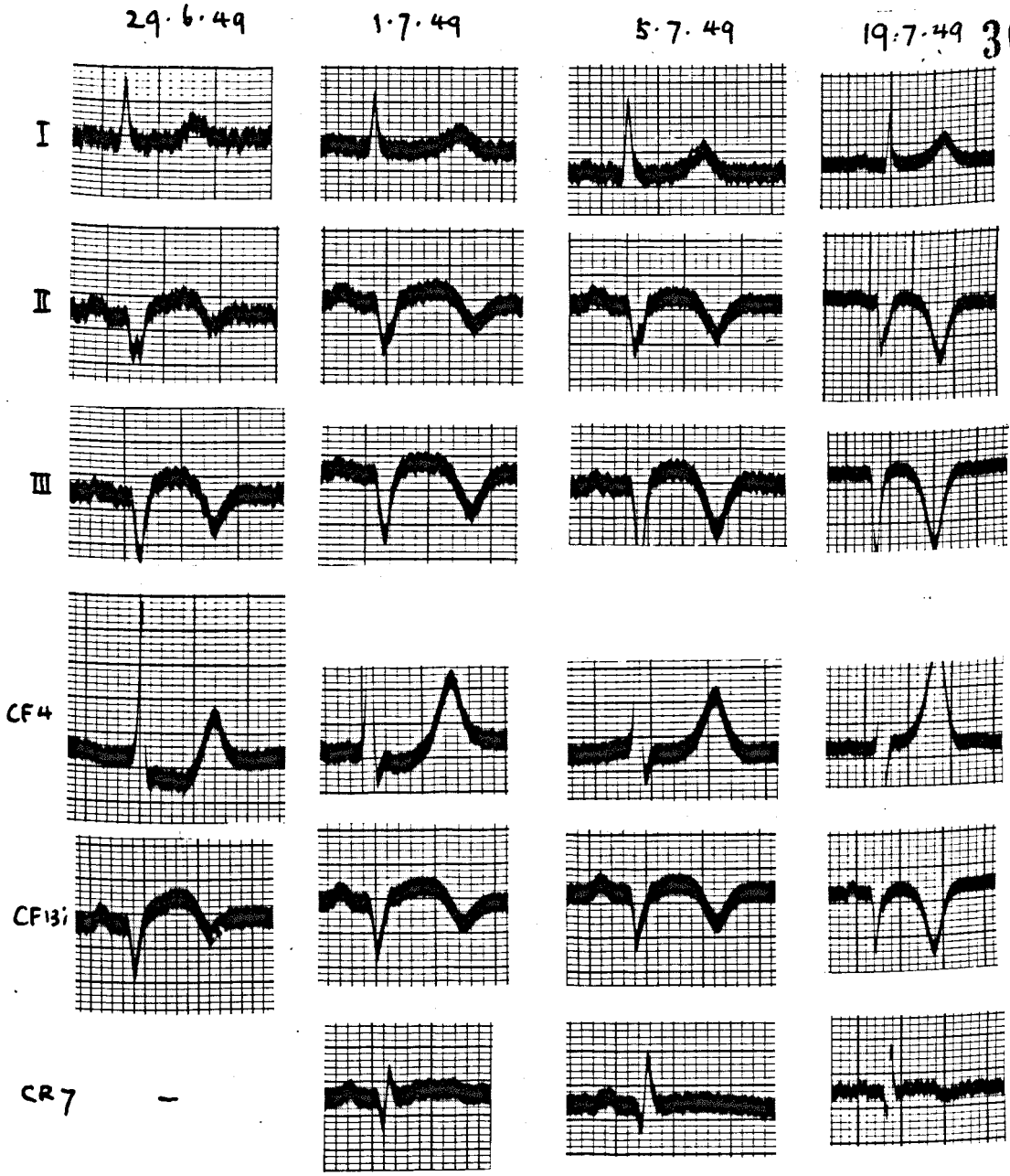


Figure 15. The evolution of CF13i and CR7 in posterior infarction.

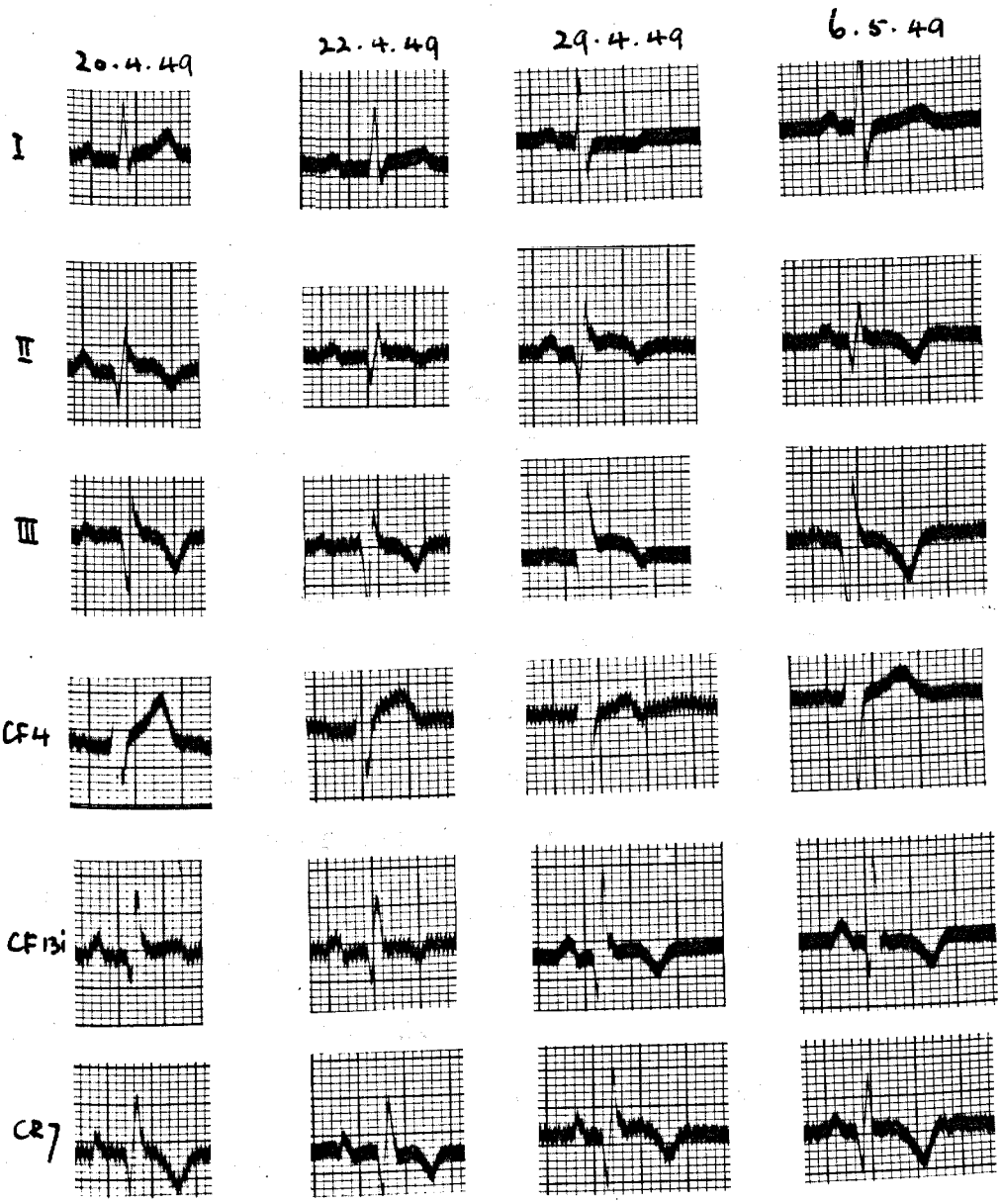


Figure 16. Another case showing the evolution of CF13i and CR7 in posterior infarction.

Variations of CF13i with heart position.

The variations of CF13i with different positions of the heart within the chest may be predicted from the changes in lead II.

A horizontal heart shows leftward deviation of the mean electrical axis of the QRS complex, resulting from the main deflections in leads I and III being directed away from each other. Lead II varies with the degree of axis deviation. If slight, the main deflection in II is upward; if more marked R and S are about equal, and if considerable the main deflection is downward.

Similarly, a vertical heart will show right axis deviation and in lead II the main deflection will be upward if it is of slight degree. If more marked R and S will be nearly equal; and if there is marked right axis deviation the main deflection will be downward.

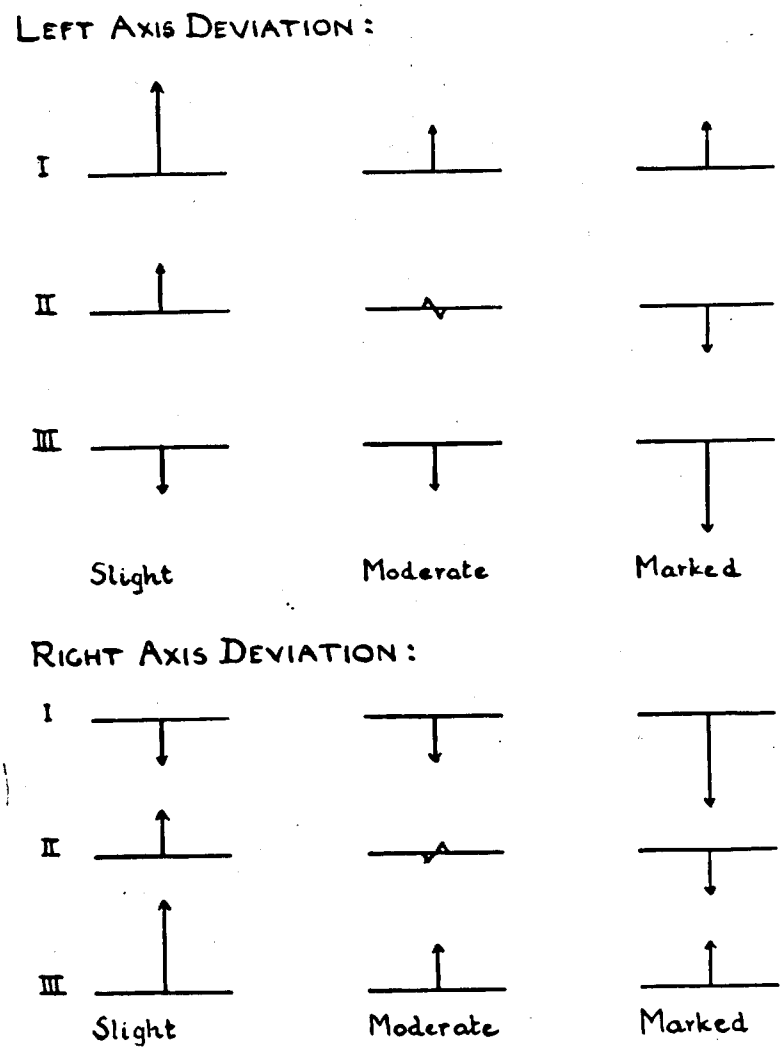


Figure 17. Varying grades of axis deviation.

It follows, therefore, that, in general, variations in lead II are independent of heart position unless this is extreme. The same may be deduced of CF13i.

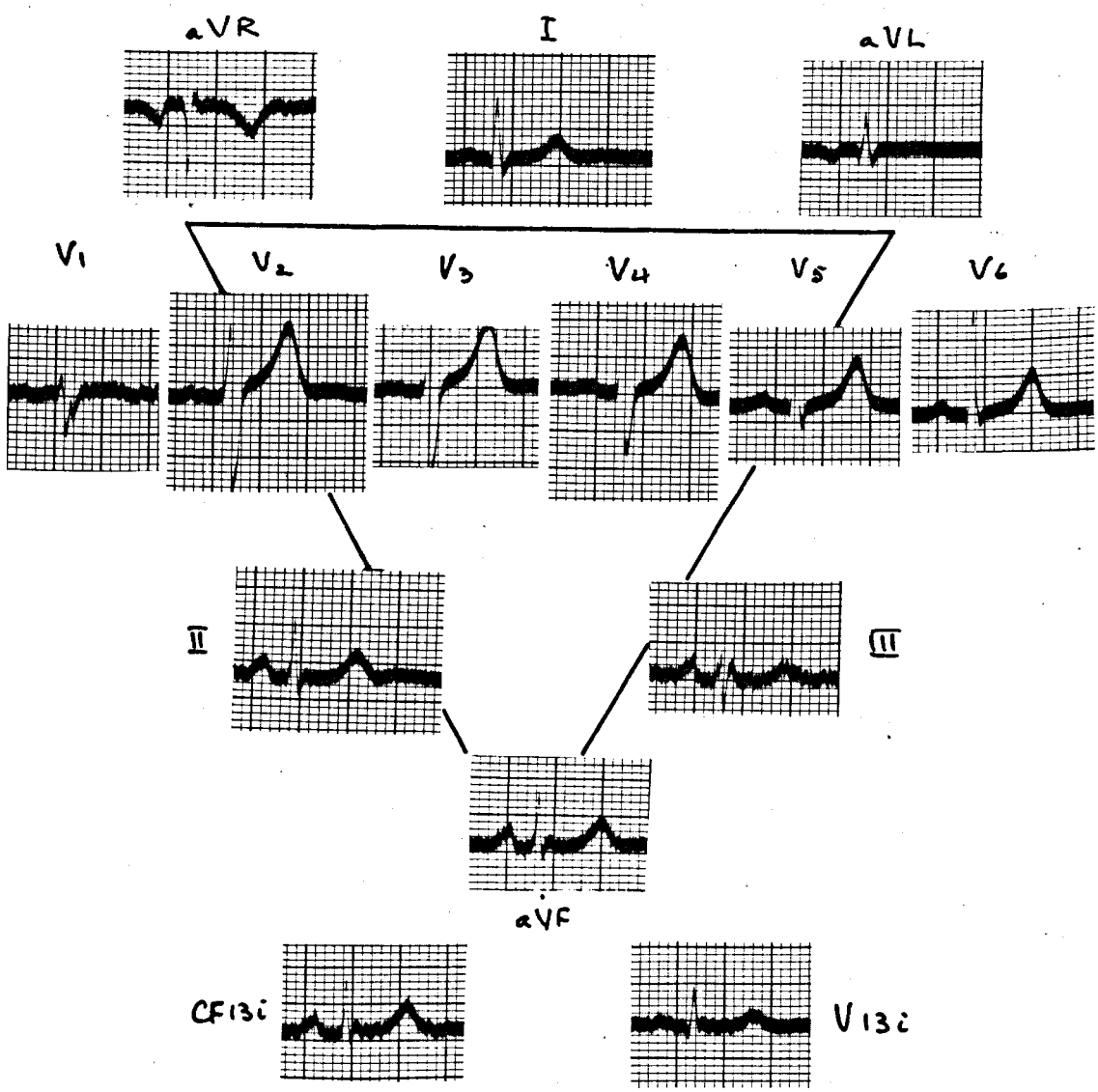


Figure 18. The heart in an intermediate position.

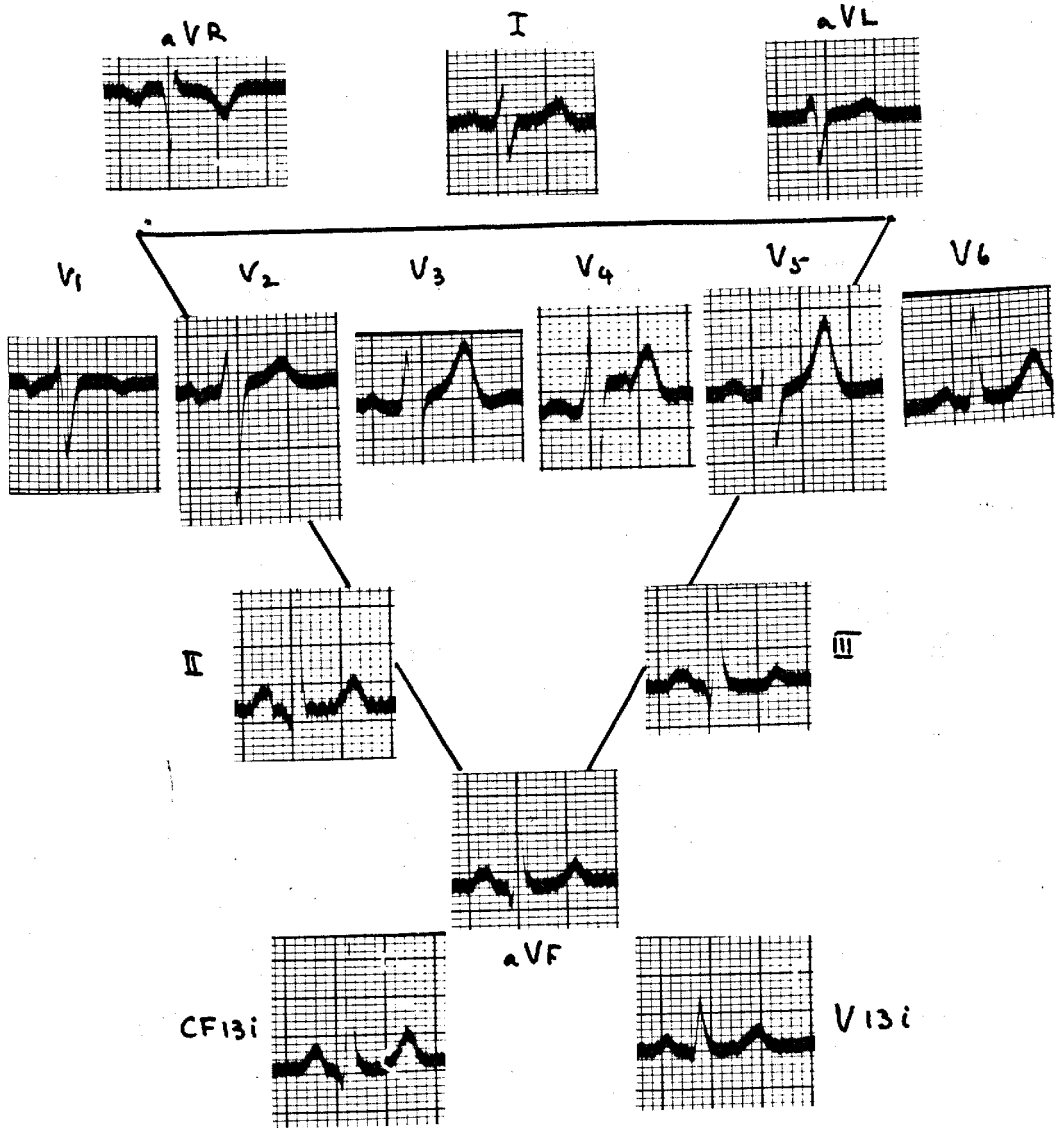


Figure 19. A vertical heart.

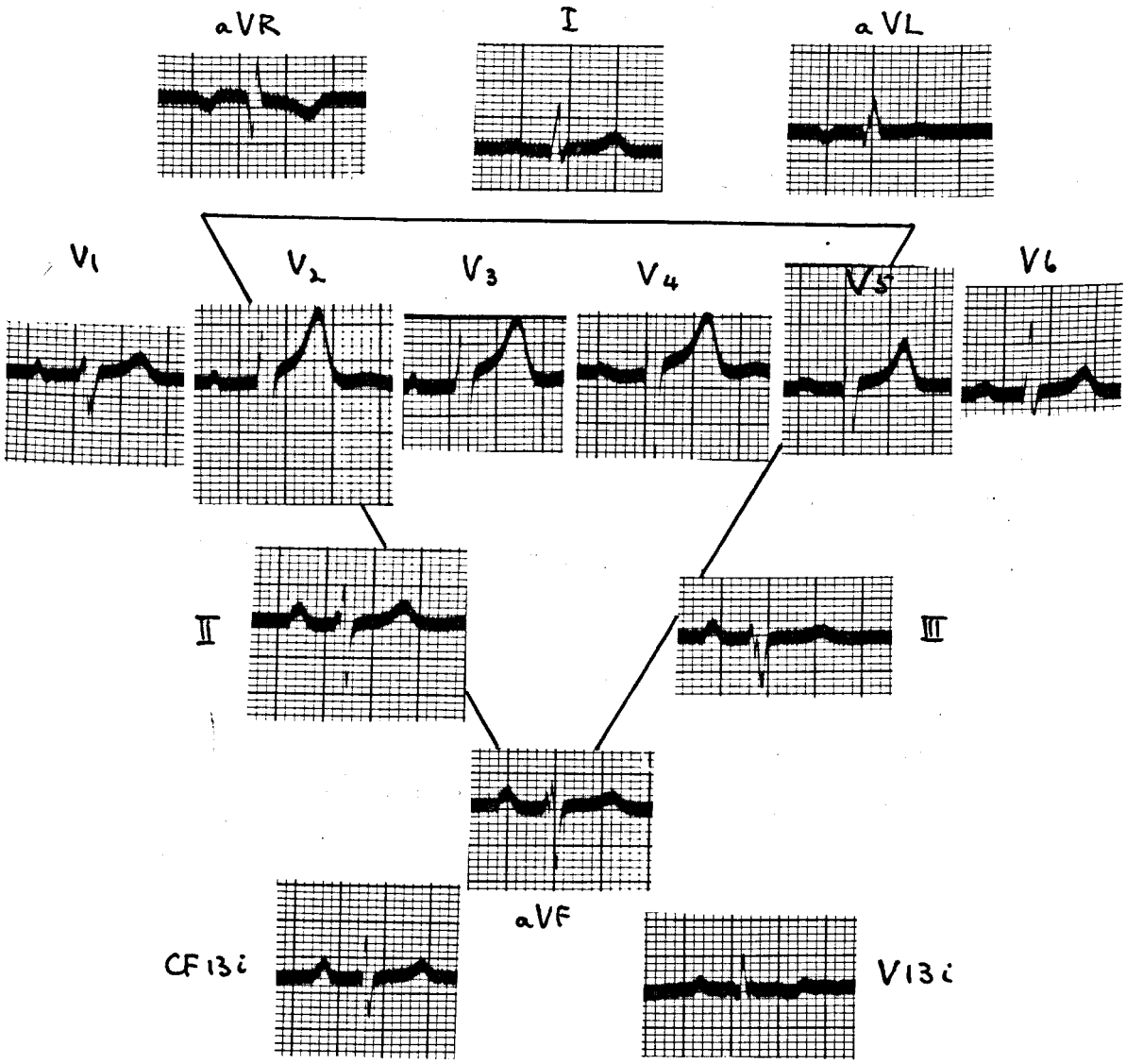


Figure 20. A horizontal heart.

Three examples are given of electrocardiograms from normal hearts showing the heart in:

1. an intermediate position (Figure 18),
2. a vertical position (Figure 19),
3. a horizontal position (Figure 20).

The vertical heart shows a slight degree of right axis deviation and CF13i shows no particular departure from the normal pattern; the horizontal heart, however, shows a marked degree of left axis deviation and in CF13i the R and S waves are nearly equal.

The next two figures are from cases of right and left ventricular hypertrophy. The former is from a case of long-standing bronchiectasis; the latter from one of hypertension.

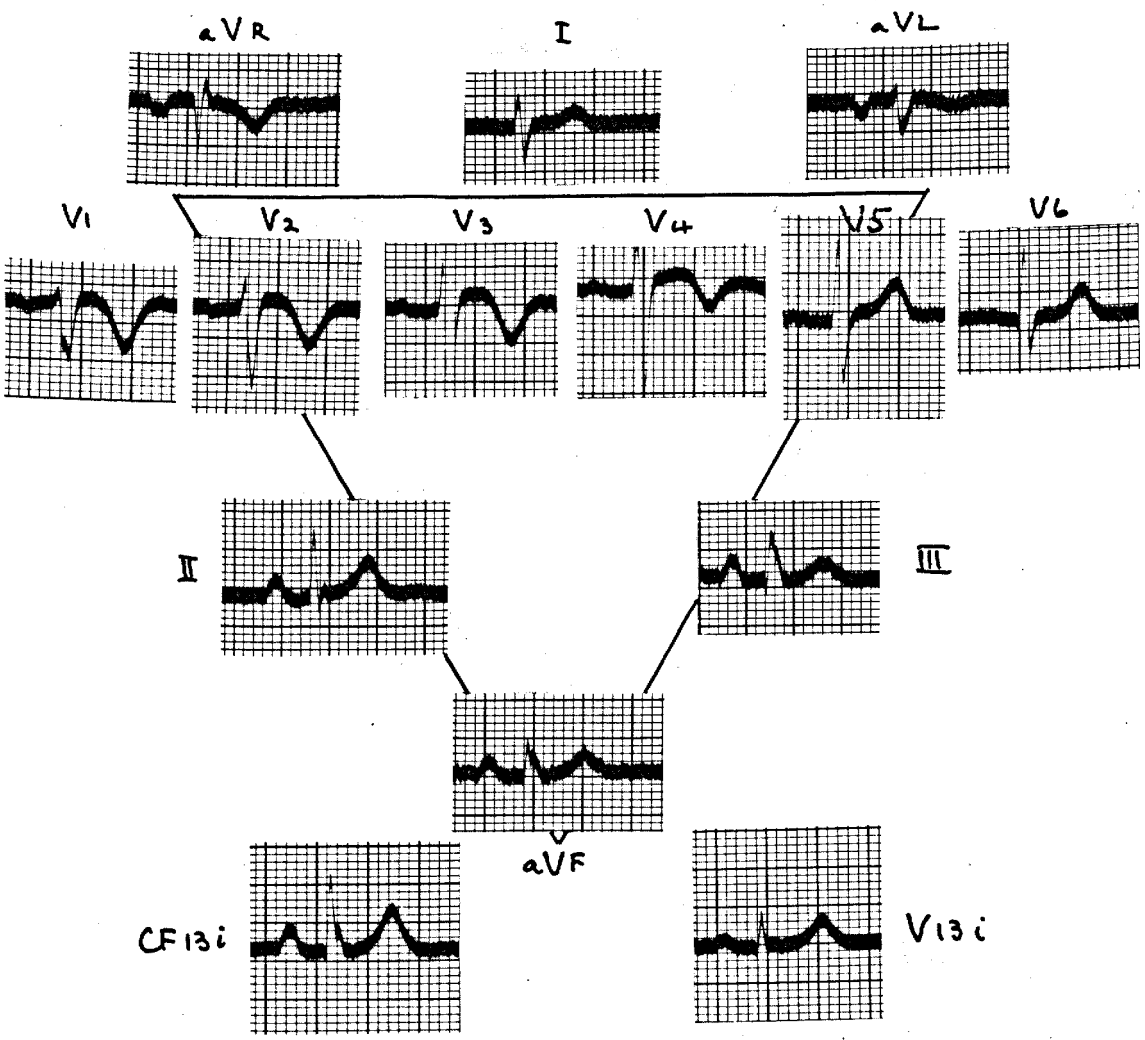


Figure 21. Right ventricular hypertrophy.

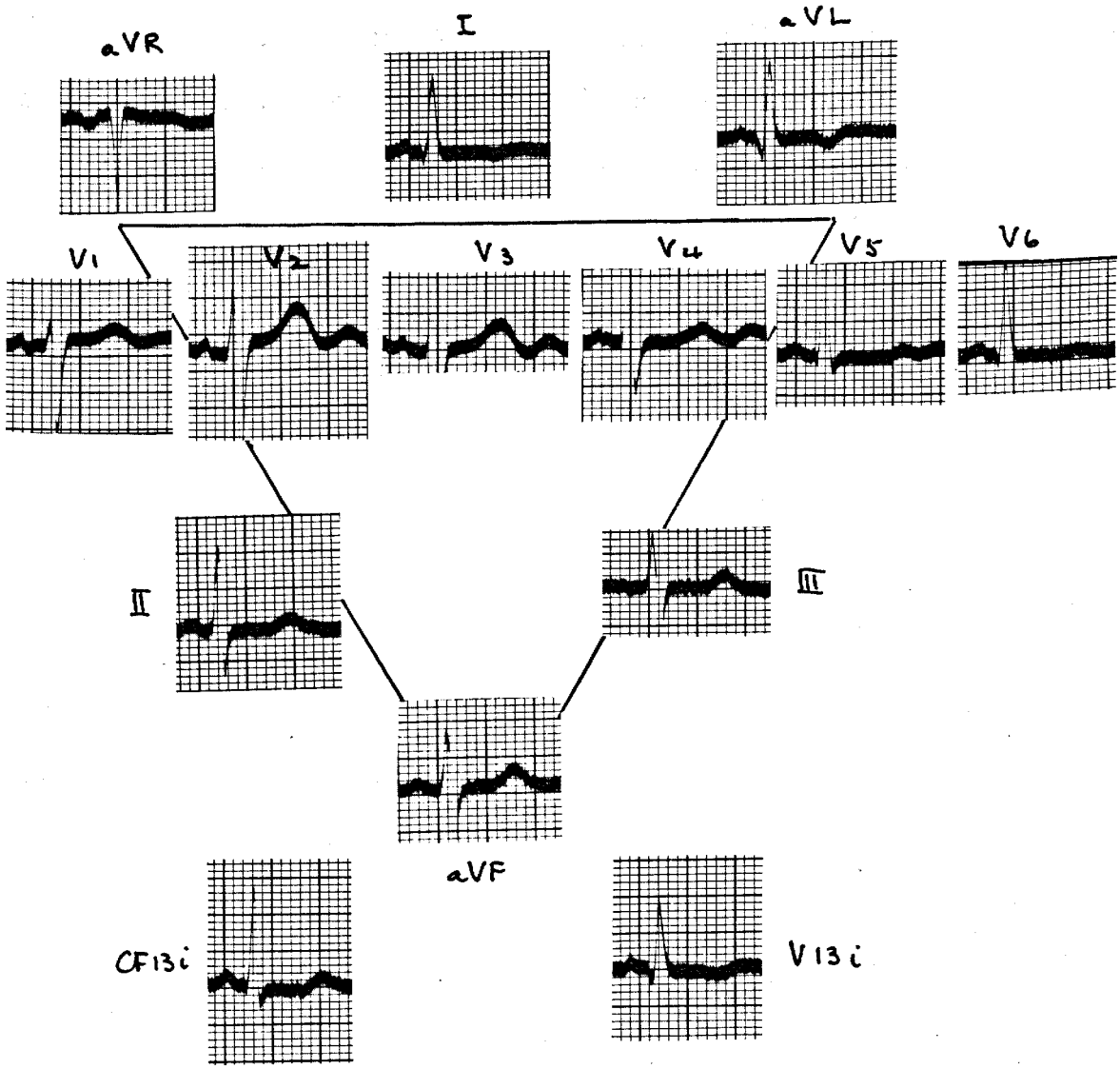


Figure 22. Left ventricular hypertrophy.

In passing, it is worth noting the following point in Figure 22. V13 may be expected to show the reverse pattern of V7 because these points are diametrically opposed (compare Figure 14). V13i therefore should resemble V7. In left ventricular hypertrophy leads over the left ventricle such as V6 and V7 show an inverted T. V13i shows the pattern expected of V7.

Correlation of E.C.G. and pathological findings.

It has been possible to obtain the heart at post-mortem examination in nine cases where at some time prior to death, electrocardiograms including CF13i had been recorded.

Most cases of myocardial infarction survive a first attack, and when death occurs there is frequently more than one infarct and usually much ischaemic fibrosis. Smith, Goodrich & Needles (1939) refer to the heart, in such circumstances, as one of ancient and multiple infarctions, with or without a superimposed, recent, single infarction. Sprague & Orgain (1935) found the same. The electrocardiogram, too, reflects the complexity of the myocardial damage.

The heart after removal from the body was fixed in formol saline. It had been hoped to inject the coronary arteries in each case with a radio-opaque mass, but for various reasons this was only possible

in one case. The method which seemed most suitable to show the site of infarcts was that of Kossman & de la Chapelle (1938; 1939a; 1939b). They cut the heart, fixed whole, into a series of sections about 1 cm. thick. These were arranged in order and photographed. The method of Schlesinger (1938) involves the injection of the coronary arteries, fixing the heart, and then "unrolling" the heart by a special procedure so that the septum is excised and the ventricular walls shown in one plane. The coronary arterial system is well shown but infarcts are not. A better method has been adopted by Myers and his colleagues in a series of papers correlating electrocardiographic findings in myocardial infarction. These papers show the results in 161 hearts collected over a period of seven years (Myers, Klein, Stofer & Hiratzka, 1947; Myers, Klein,& Stofer, 1948; Myers, Klein & Hiratzka, 1948; Myers, Klein & Stofer, 1949; Myers, Klein & Hiratzka, 1949a, 1949b, 1949c, 1949d). They utilised Schlesinger's method at first but abandoned it in favour of a modified Kossmann - de la Chapelle technique in which the coronary arteries were injected with a radio-opaque mass, fixed, and then

cut into serial slices. These were arranged in order and then radiographed. That this method is not entirely satisfactory is shown by their having to outline the infarcts in the radiographs with a grease pencil from naked eye inspection of the serial slices. One point, which is well brought out, is the very irregular shape of most infarcts. The simple division of infarcts into anterior, lateral and posterior is an over-simplification, which their work serves to prove.

One case in this series has been prepared by Myers' method, and the radiographs are shown. They compare unfavourably with photographs of the sections.

Since this work was begun, another method has been described (Sheldon, Sayen, 1949; Sayen, Sheldon, 1949). The second of these papers is entitled "Difficulties of description and illustration of ventricular muscle lesions ..." and draws attention to the defects in methods previously used. It describes the construction of a "myocardial map". The left ventricle is shown in a simple diagram split into three parts. The extent of the infarct is determined by the method of serial slices and indicated

on the diagram. One disadvantage is that infarcts encroaching into the right ventricle from the left cannot easily be shown. Altogether the Kossmann - de la Chapelle technique would appear to be the best.

Case No. 1.

H.G., a labourer, aged 54, was seen at an Out-patient Clinic 11th October 1948. He had been complaining of a gripping substernal pain on effort for three weeks. There were no previous illnesses. Physical examination revealed congested neck veins, scattered rhonchi in the chest, slight ankle oedema and hepatic enlargement. The pulse showed an irregularity due to premature beats, but there was no clinical cardiac enlargement. The heart sounds were normal and there were no murmurs. Blood pressure 125/?.

An x-ray of the chest that day (Figure 23, A, B & C) was reported as follows:-

The lung fields show quite considerable congestive changes superimposed on general pulmonary fibrosis. There is considerable cardiac enlargement in all diameters but predominantly left ventricular in type. Screening showed no evidence of aneurysmal dilatation of the aorta.

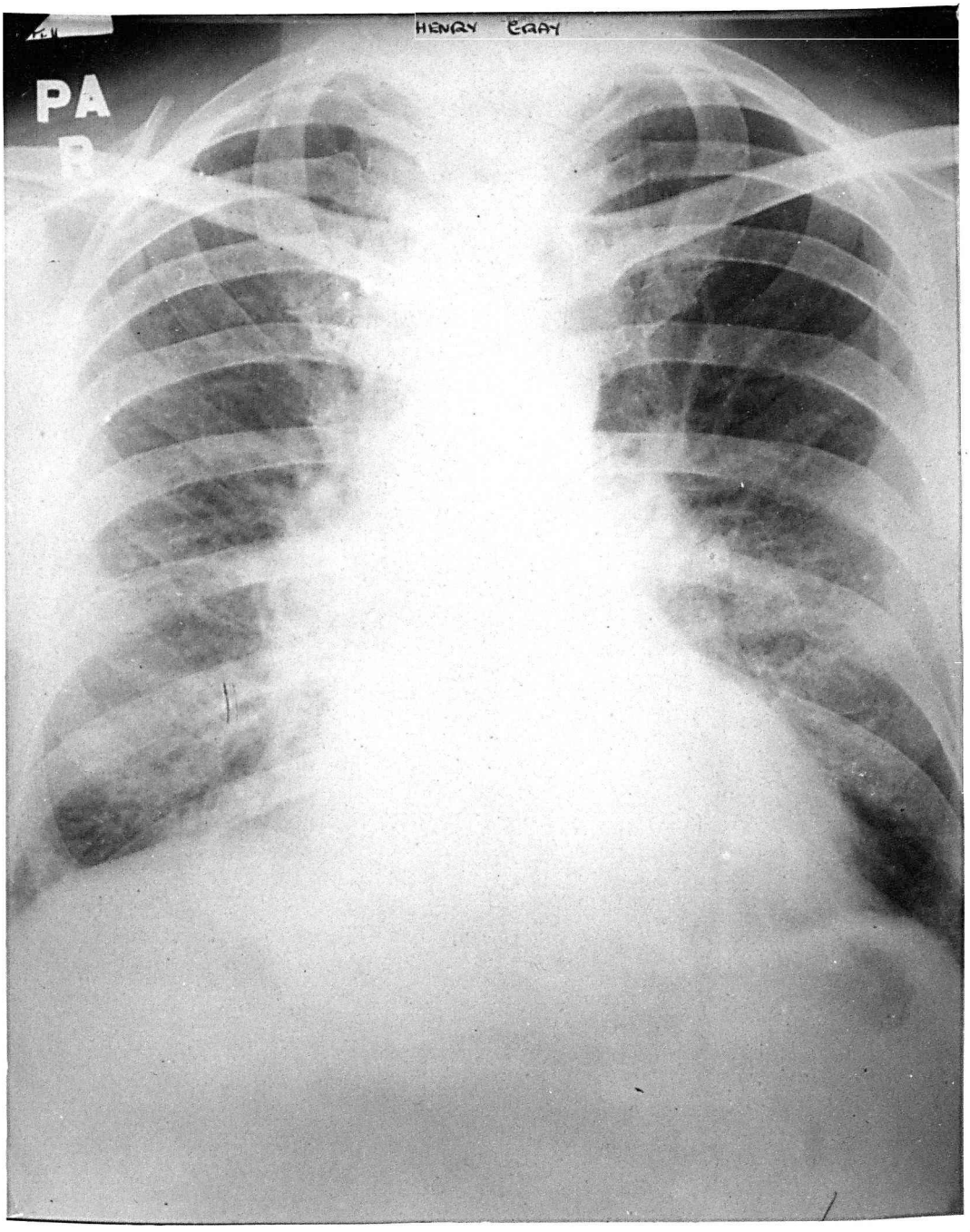


Figure 23 A. Case No.1. H.G. Postero-anterior view of the chest.

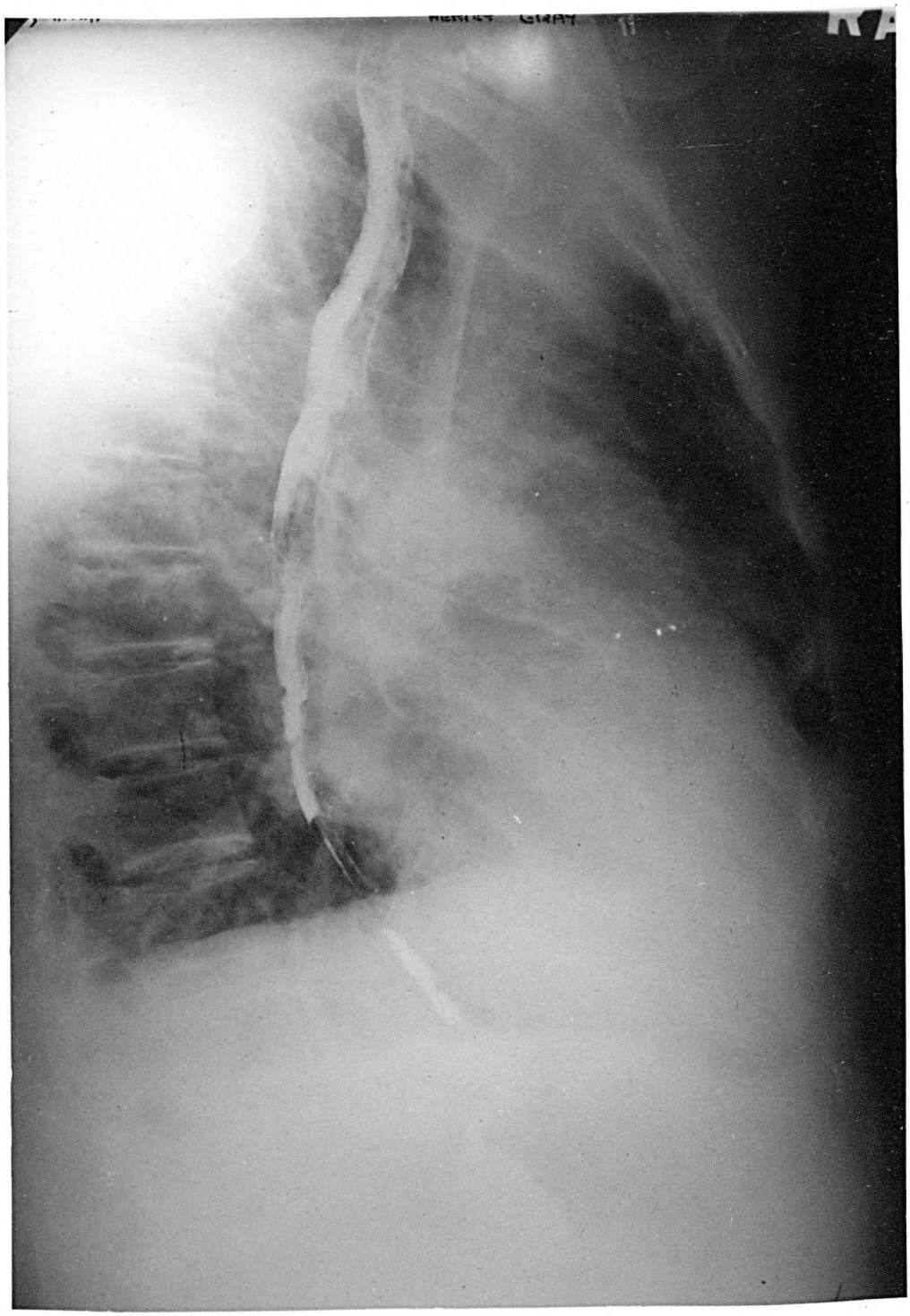


Figure 23B. Case No. 1. H.G. Right oblique view of the chest.

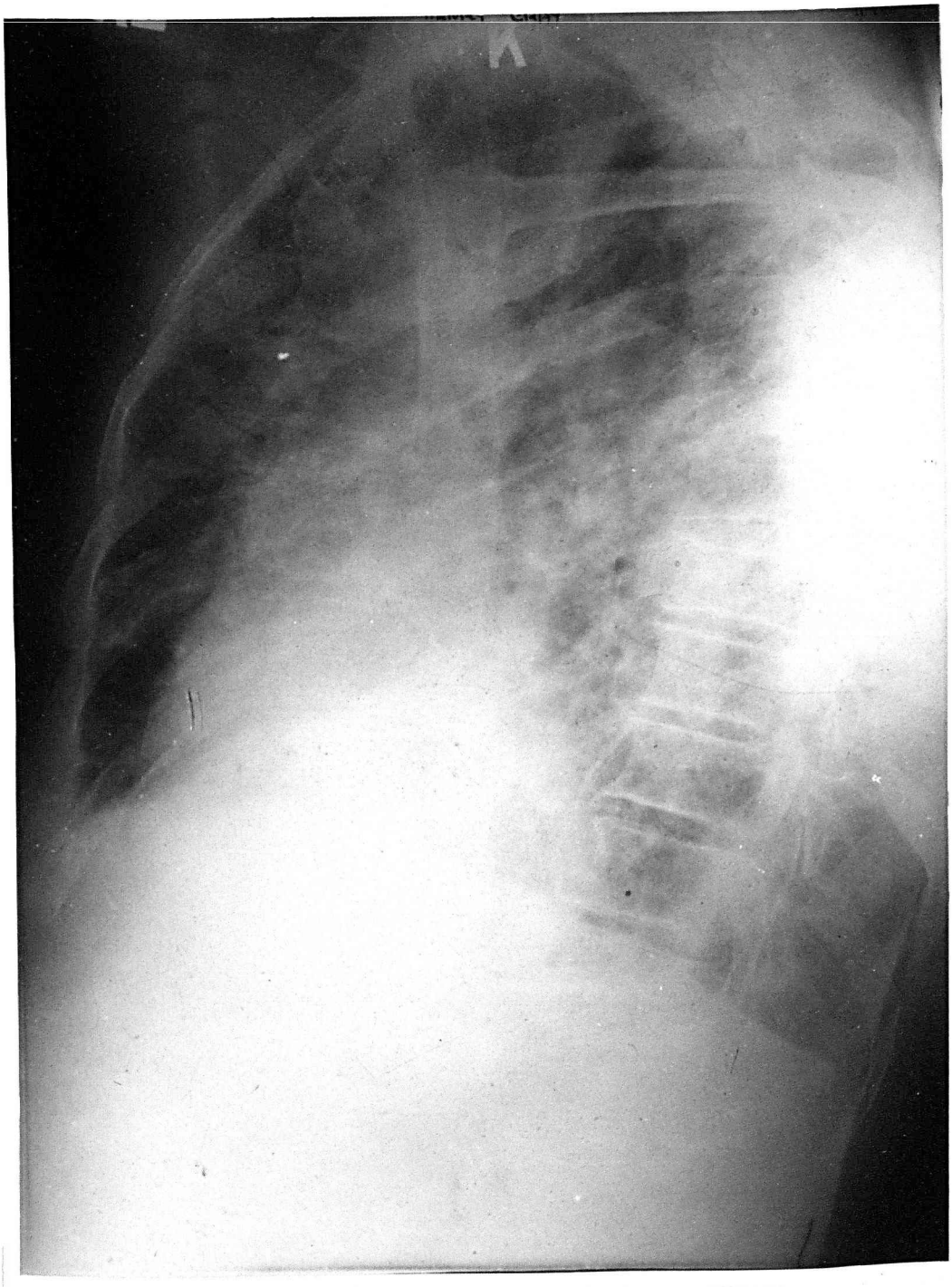


Figure 23 C. Case No. 1. H.G. Left oblique view of the chest.

An electrocardiogram taken on 15th October 1948 showed sinus rhythm with auricular premature beats. There was ST depression in lead I and elevation in lead III with T wave inversion in leads II and III (Figure 24).

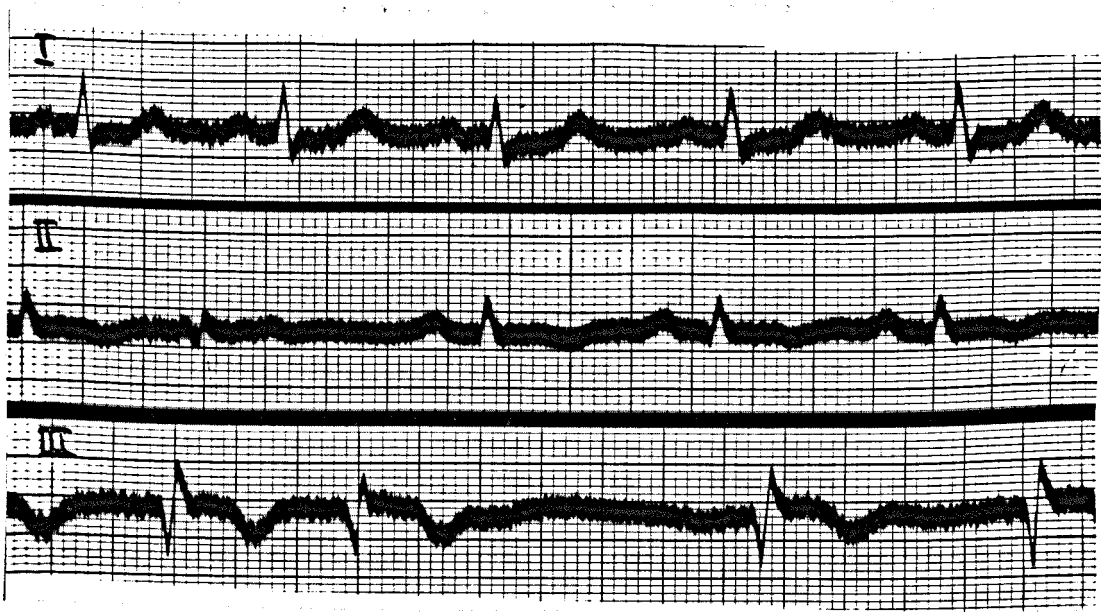


Figure 24. Case No. 1. H.G.
Electrocardiogram 15.10.1948

The patient was admitted to hospital on 15th October 1948. His condition gradually deteriorated. The oedema increased and ascites developed despite treatment, and he died 10th November 1948.

The blood Wassermann reaction was negative.

A further electrocardiogram was taken 20th October 1948 including leads CF4 and CF13. The latter has been printed showing reversed polarity. The standard leads show no change. The apical lead shows a deep QS complex. CF13i shows an inverted T wave, thus amplifying the T wave change of standard lead II (Figure 25).

Figure 25. Second electrocardiographic record.

Autopsy.

There was free fluid in the peritoneal and pleural cavities, and marked chronic venous congestion of the liver. The lungs were emphysematous with large bullae on the anterior margins, and showed brown pigmentation.

The heart weighed 445 grams. There was an area of aneurysmal thinning in the left ventricle

posteriorly near the apex. The aorta showed moderate atheroma.

Serial slices of the heart after fixation are shown in Figure 26. ✠

Section 1 shows a fibrous band in the posterior part of the septum extending into the posterior wall. Section 2 shows thickened endocardium in the posterior part of the septum and posterior wall of the left ventricle. Beneath there is dense fibrous tissue. In the anterior part of the septum are a few patchy areas of fibrosis.

✠ The following convention has been adopted for displaying the sections of the heart in this and subsequent cases. The topmost section is shown in the top left hand corner of the photograph. Succeeding sections are shown below it and continue in the top right hand corner and below that. The top edge of each section is the posterior surface. The right ventricle is therefore towards the left hand edge of the photograph: the left ventricle towards the right hand edge. The measures are marked in millimetres and centimetres.

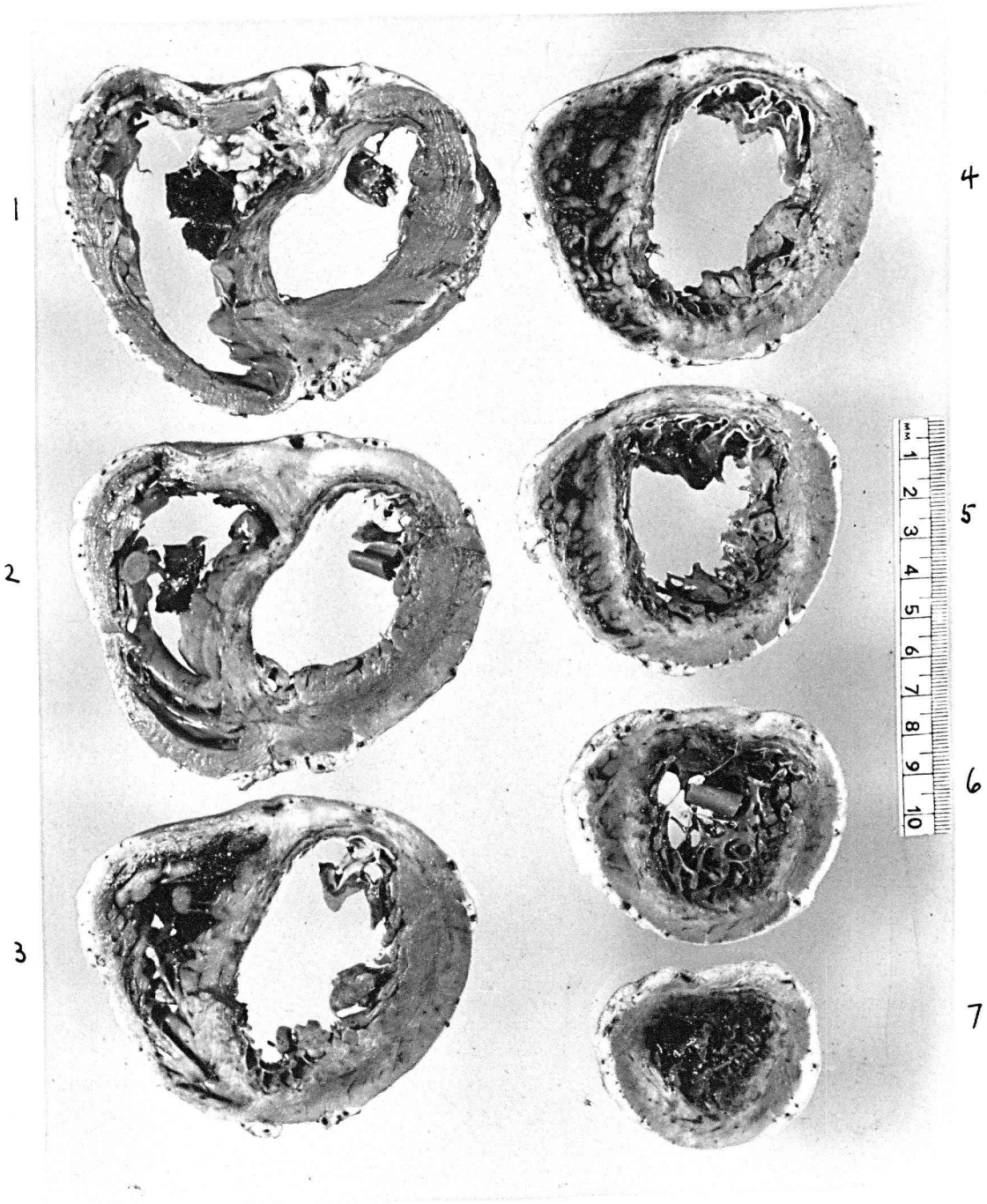


Figure 26. Case No. 1. H.G. Serial slices of the heart.

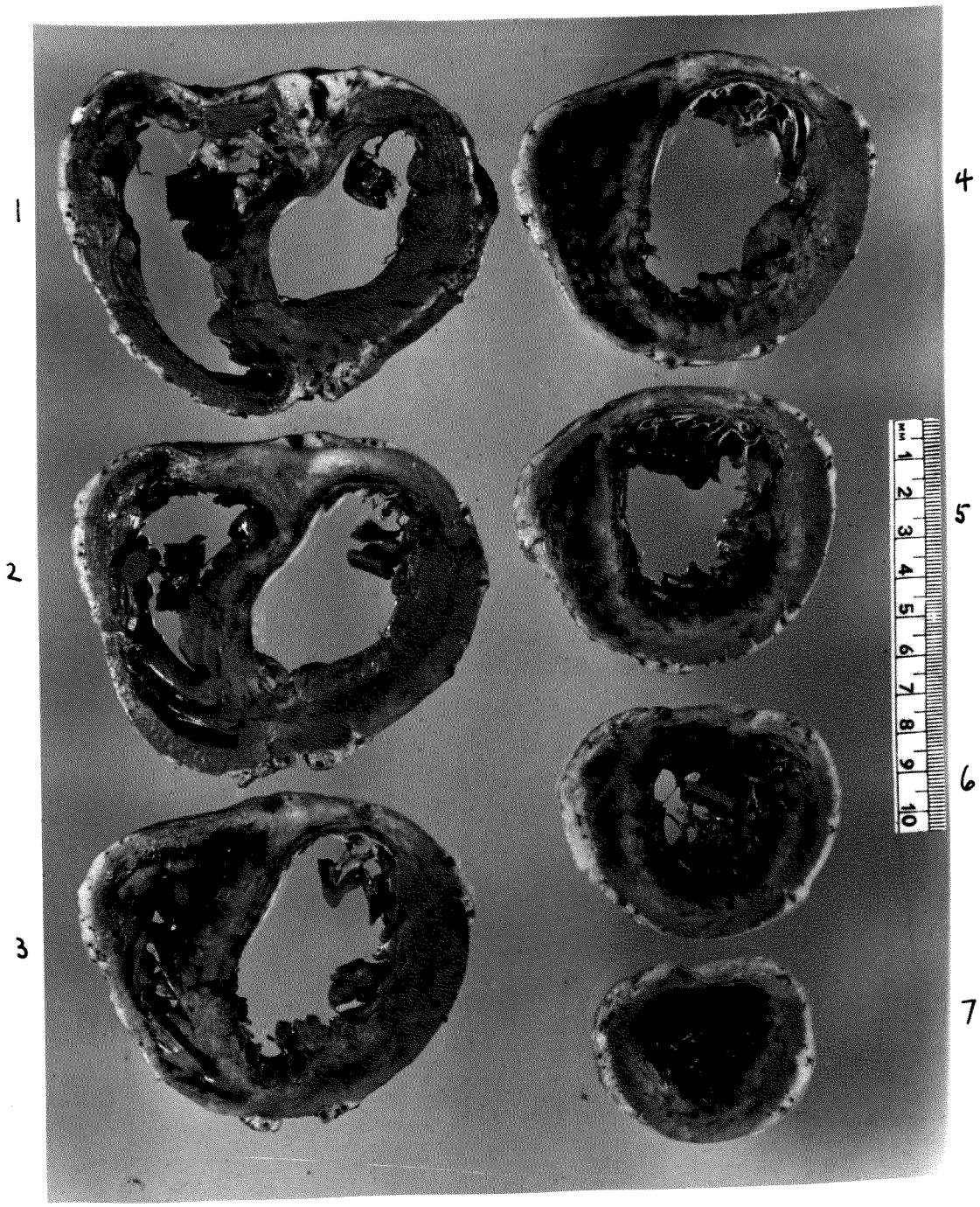


Figure 26. Case No. 1. H.G. Serial slices of the heart.

Section 3 shows a similar picture with fibrosis extending into the right ventricle posteriorly. Section 4 is shown enlarged in Figure 27. The posterior infarct is well shown and it extends into the right ventricle and the septum. It also extends round subendocardially into the anterolateral area of the left ventricle.

Section 5 shows the infarcted area encircling the whole of the left ventricle subendocardially and similar appearances are shown in sections 6 and 7.

Summary.

Three weeks' effort angina followed by congestive cardiac failure. Electrocardiographic examination shows evidence of anterior and posterior infarction confirmed by autopsy evidence but the posterior lesion predominates. The age of the infarct from the clinical data was about eight weeks and the macroscopic appearances are compatible (Mallory, White & Salcedo-Salgar, 1939).

Comment.

The infarct is cup-shaped, the cup corresponding to the apex of the left ventricle. A tongue is

Figure 27. Section 4 of Figure 26, enlarged.

prolonged upwards posteriorly in the left ventricle and also into the right.

The standard lead electrocardiogram shows no evidence of anterior infarction. The apical lead displays it: and CF13i amplifies the evidence of the standard leads.

Case No. 2.

F.Y., a football pool supervisor aged 61, attended the Heart Clinic 10th November 1948. Three nights previously, in bed, he was awakened by a severe substernal pain, which radiated across the chest to the right shoulder. He was dyspnoeic also. He got out of bed, sat in a chair, and shortly after vomited. The pain continued several hours.

Twelve years previously he had had a similar attack for which he was treated in hospital for five weeks. No details of this could be obtained. There had been occasional attacks of pain on effort in the interim. There were no other significant previous illnesses.

On examination, the patient was seen to be dyspnoeic at rest and he had a persistent tachycardia. The heart sounds were normal. Blood pressure 100/75. Urine normal.

An electrocardiogram (Figure 28) showed sinus tachycardia, low voltage of the standard leads and left axis deviation. T_2 was barely inverted but T_3 was negative. CF4 showed slight ST depression.

CF13i showed a very small amplitude of the QRS complex but it was quite plain that the T wave was inverted.

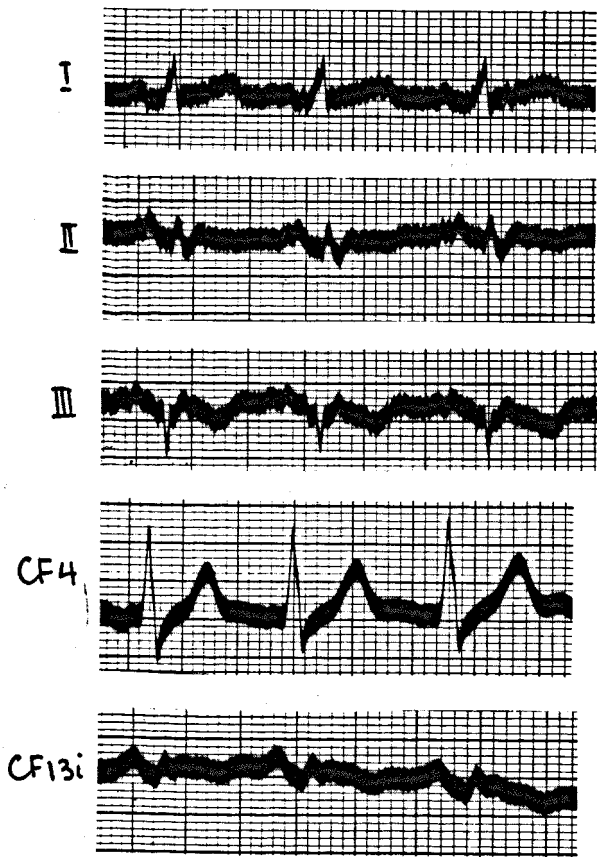


Figure 28. Case No. 2. F.Y.
Electrocardiogram 10.11.1948

The patient was admitted straight away. He continued to have pain, became collapsed and the blood pressure fell to 90/70. Two days later a further electrocardiogram showed reduced voltage of the standard leads and Q_3 became evident. Q_2 did not show but CF13i showed a broad Q. (Figure 29).

Ten days after admission the patient was in extremis and the blood pressure could not be recorded. Four days later he died.

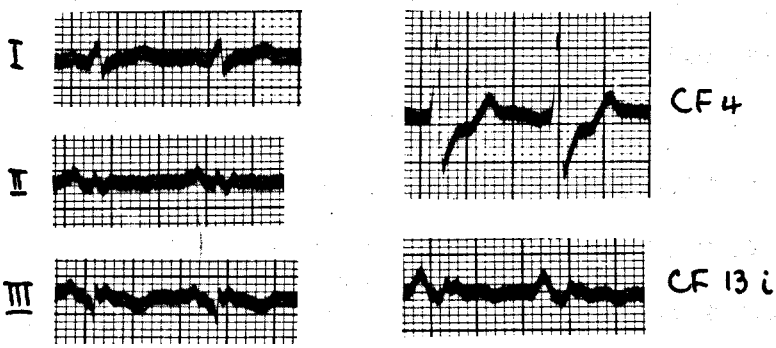


Figure 29. Case No. 2. F.Y.
Electrocardiogram 12.11.1948

Autopsy.

Apart from the heart there was little of note. The liver was congested and the lung bases showed patchy consolidation. The renal and cerebral arteries showed atheroma.

The heart weighed 380 grams. An occlusion in the circumflex branch of the left coronary artery

was noted. There was an old dense fibrous scar at the apex of the left ventricle involving the adjacent part of the septum. In the lateral aspect of the left ventricle there was a yellowish area of infarction. There was no pericarditis.

The serial slices are shown in Figure 30. Section 1 shows a recent lateral infarct of the left ventricle. It extends into the anterior and posterior walls, and is yellowish in colour. Section 2 shows the recent lateral infarct and in the posterior wall of the left ventricle is a narrow band of old infarct running into the septum. The overlying endocardium is thickened. Section 3 is similar, the recent infarct being seen to extend posteriorly as far as the old. There is patchy fibrosis in the septum. Section 4 shows a similar picture. The recent character of the lateral infarct is well seen in the original from its yellow colour. Section 5 is shown enlarged in Figure 31. There is patchy fibrosis in the septum with endocardial thickening. The lateral infarct may be seen to extend posteriorly and the borders show some hyperaemia. Sections 6 and 7 show dense fibrous tissue capping the apex and the left side of the septum. The recent infarct may be seen postero-laterally.

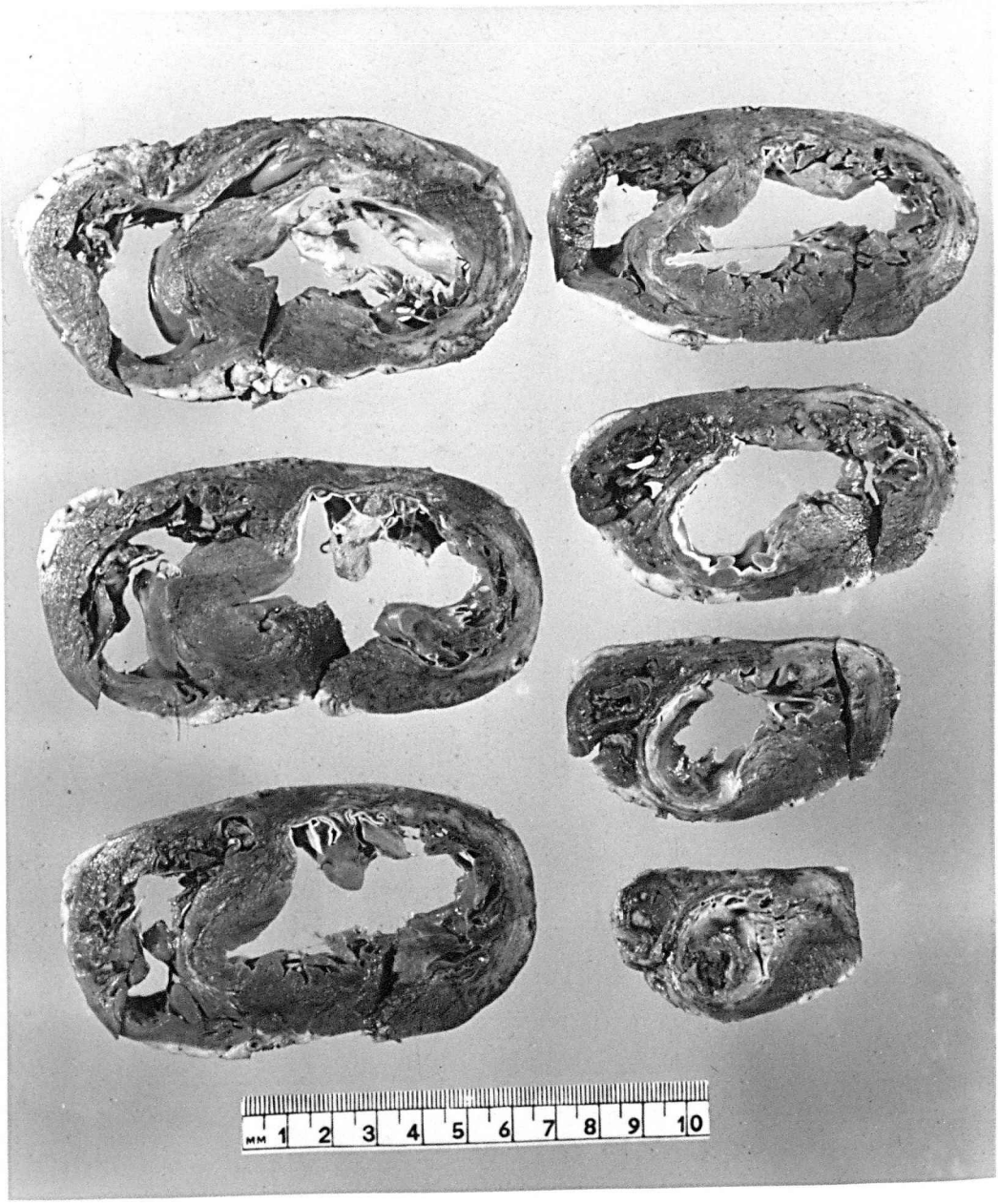


Figure 30. Case No. 2. F.Y. Serial slices of the heart.

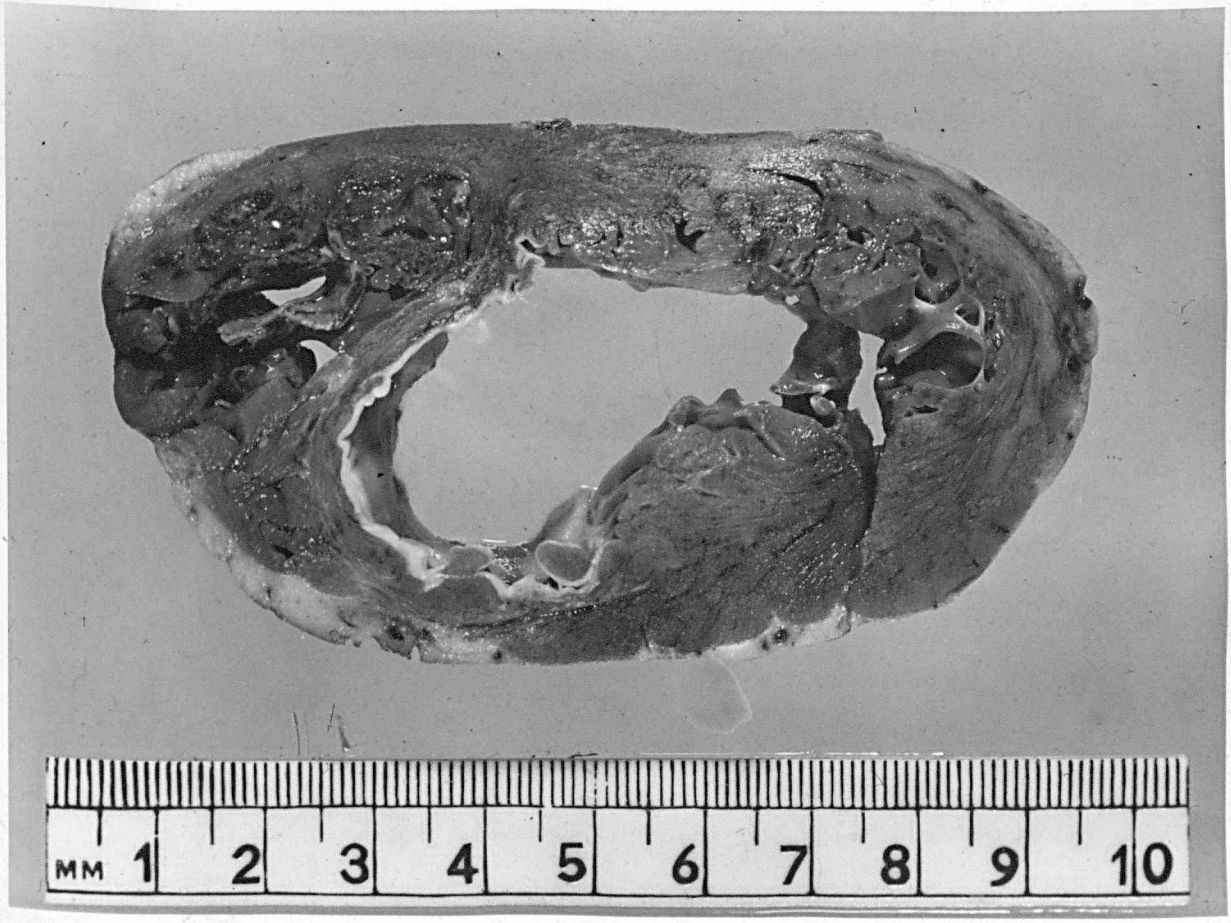


Figure 31. Case No. 2. F.Y. Section 5 of Figure 30, enlarged.

Case No. 3.

T.McG., a local government officer aged 55 attended an Out-patient Clinic in March 1948.

Apart from diphtheria in childhood he had always been well, until August 1944. He had then a sudden attack of substernal pain which lasted about five hours. It had been very violent for ten minutes, and it made him breathless. Following this episode he had been subject to pain in the chest on exertion. On 15th March 1948 a further attack of severe pain occurred at rest. It lasted about 24 hours. The patient was kept in bed one week and then sent to the Out-patient Clinic (23rd March 1948). He was admitted.

Examination showed an obese elderly man. The heart sounds were of poor quality but there were no murmurs. Blood pressure ? 160/80 in the clinic: 115/80 in the ward. The urine was normal.

An electrocardiogram taken the following day showed the Q_1 T_1 pattern of anterior infarction (Figure 32). CF13 was not recorded.

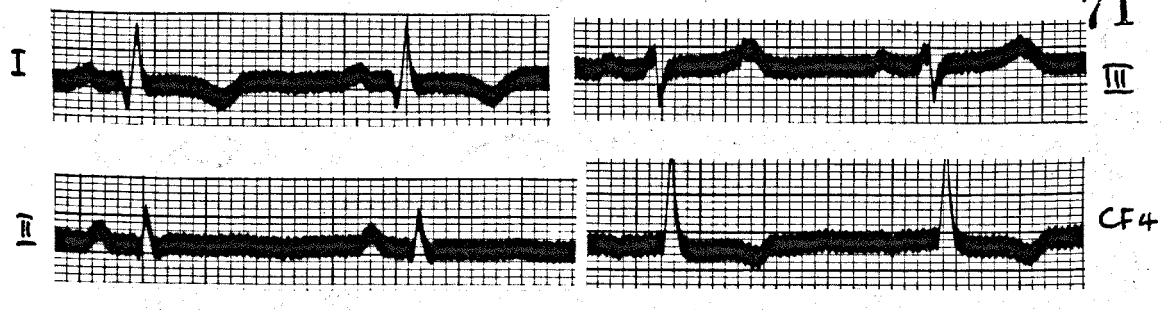


Figure 32. Case No. 3. T.McG. 24.3.1948.

The Wassermann reaction was negative, and the blood picture was normal. After four weeks strict bed rest the patient was discharged and spent his convalescence at home.

An electrocardiogram was now showing a deeply negative T_1 and negative T_2 also (Figure 33).

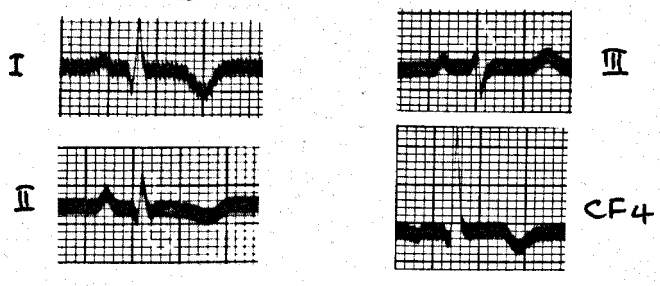


Figure 33. Case No. 3. T.McG.
Electrocardiogram 9.4.48.

He was seen again in July 1948 and, although easily breathless on effort, was feeling well. Blood pressure 160/110. No evidence of congestive failure. An x-ray of the chest (6.7.48) (Figure 34 A, B & C) showed "moderate general cardiac enlargement with left ventricular enlargement predominating: unfolding of

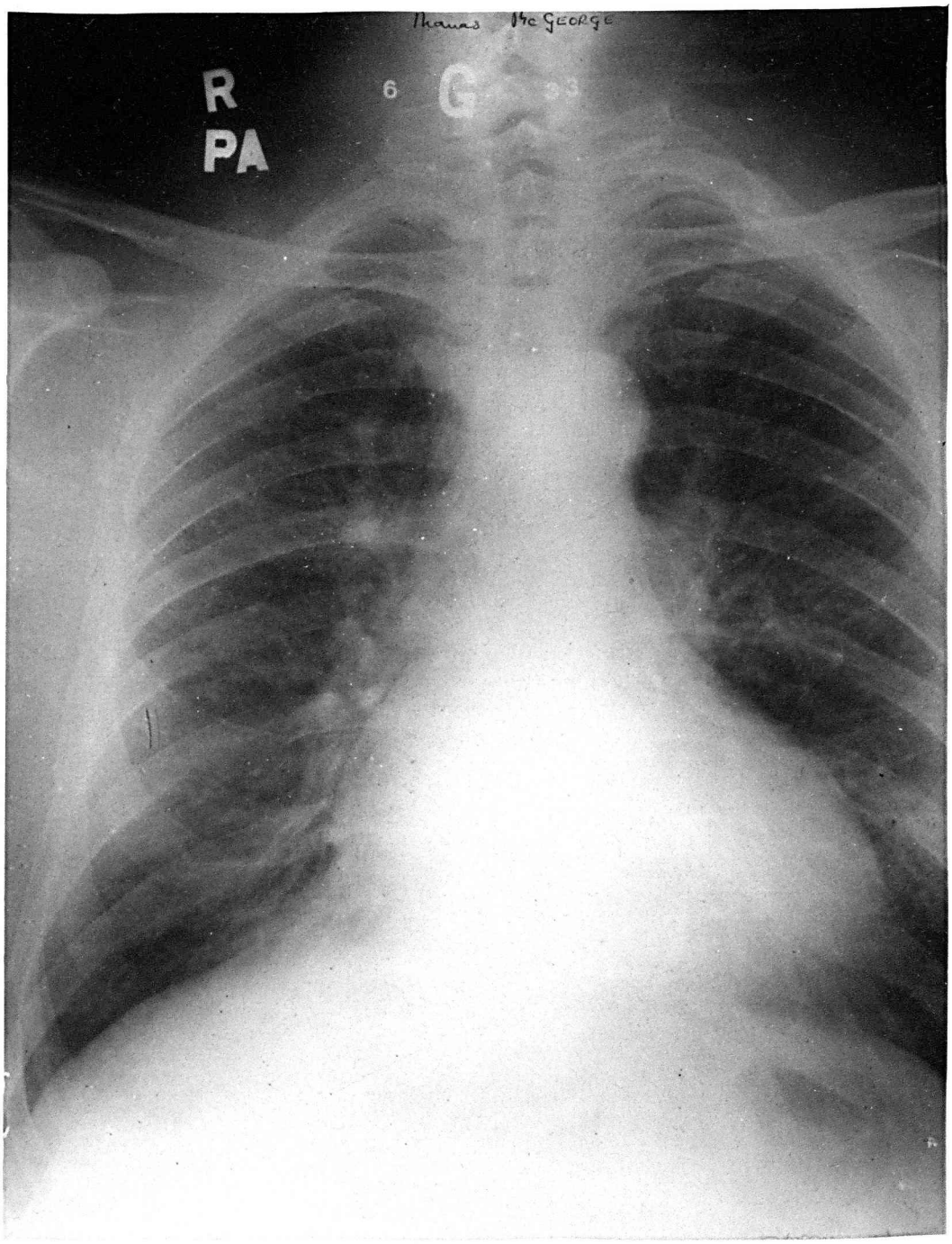


Figure 34 A. Case No. 3. T.McG.
Postero-anterior view of chest.

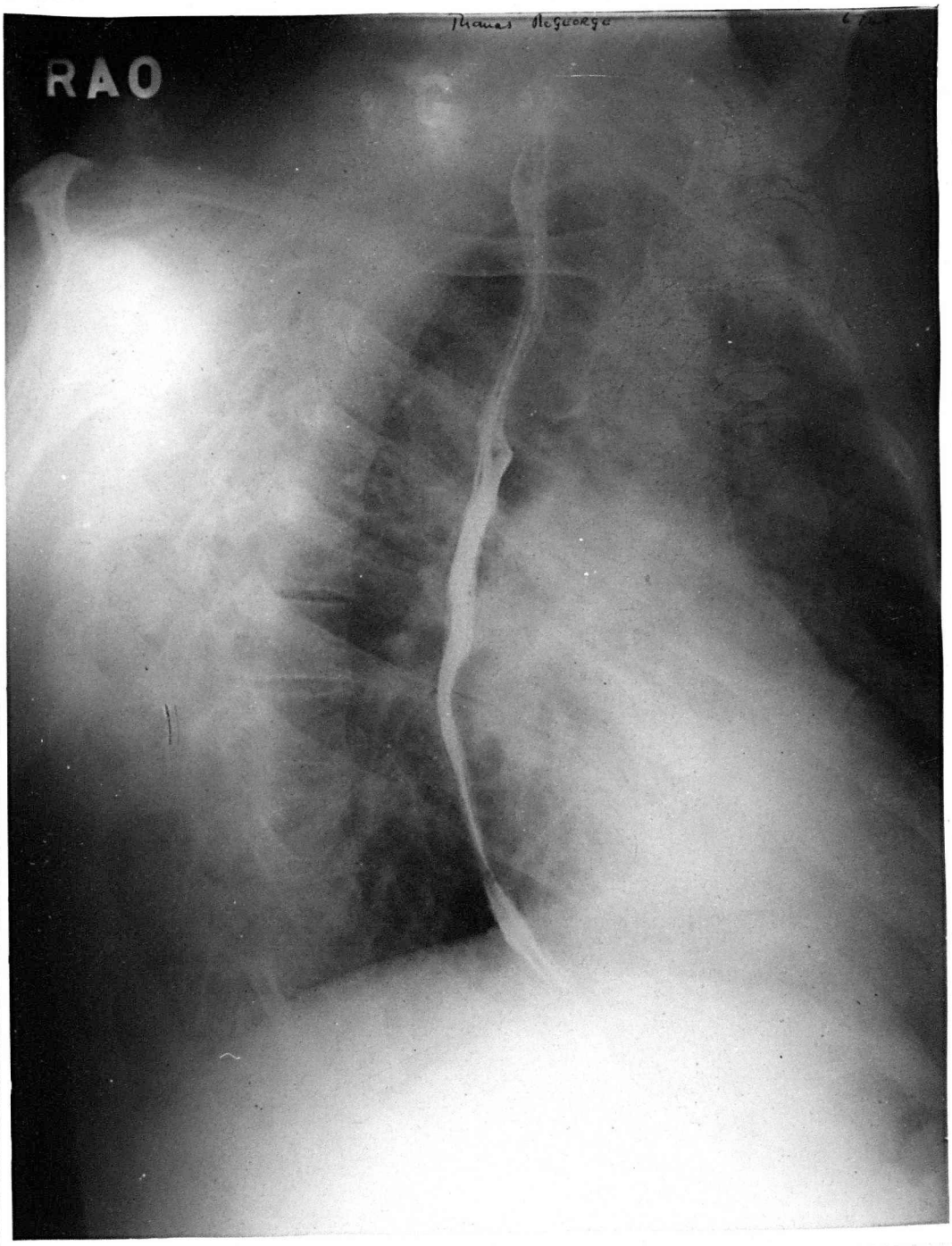


Figure 34 B. Case No. 3. T.McG.
RAO view of chest.

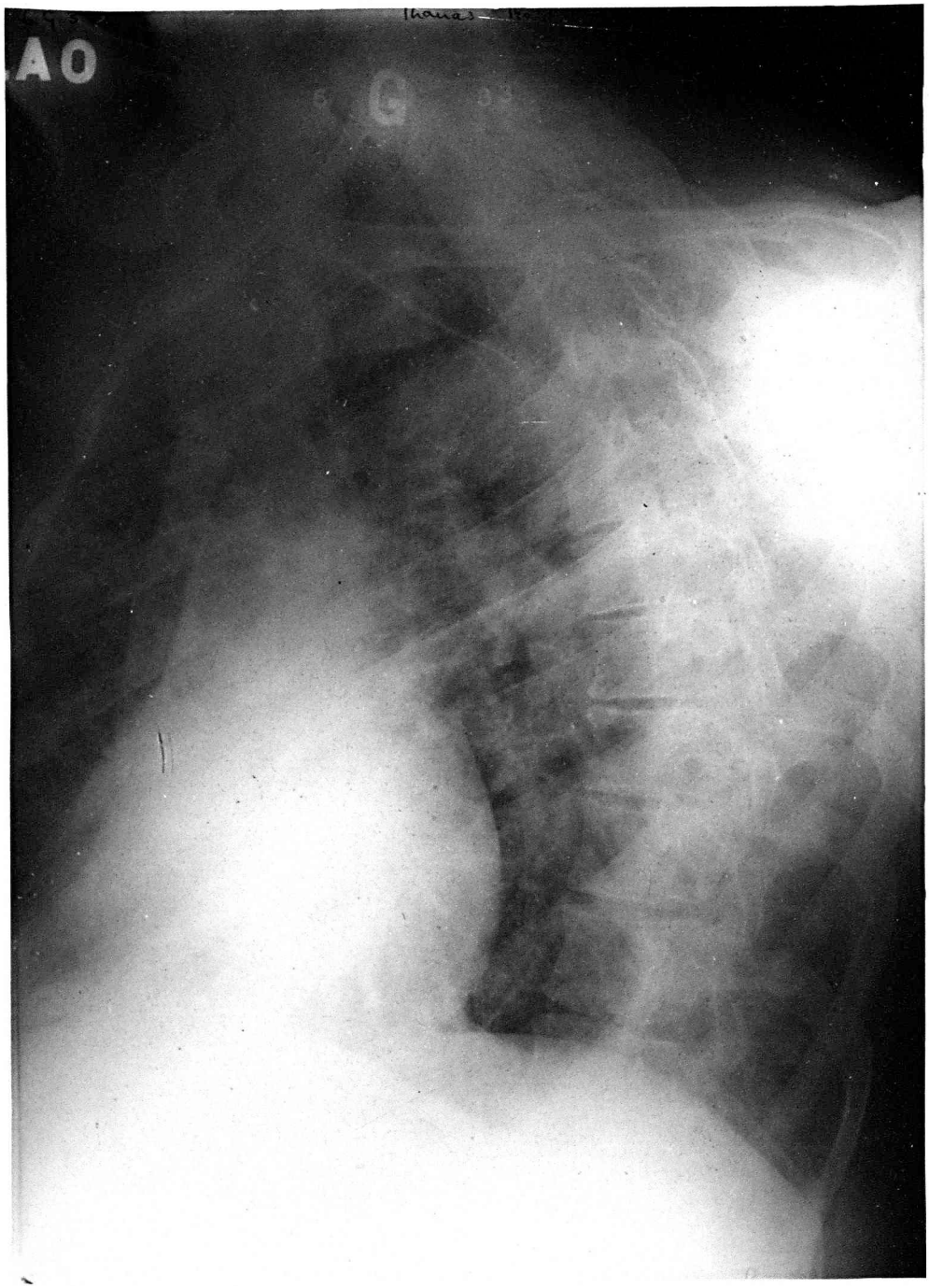


Figure 34 C. Case No. 3. T.McG.
LAO view of chest.

the aorta with atheroma (i.e. calcific flecks in the aorta.)"

The electrocardiogram (7.7.48) showed T_2 to be upright (Figure 35).

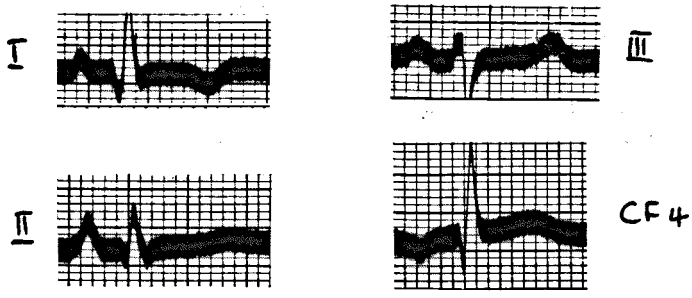


Figure 35. Case No. 3. T.McG.
Electrocardiogram 7.7.1948.

The patient was declared fit to resume his work (which was sedentary).

In January 1949, he was still easily dyspnoeic and occasionally experiencing effort pain, but managing his work quite well.

An electrocardiogram with CF13i was taken and is shown in Figure 36. The standard leads show evidence of an anterior infarct but T_2 is quite flat. It will be observed that T in CF13i is deeply inverted.

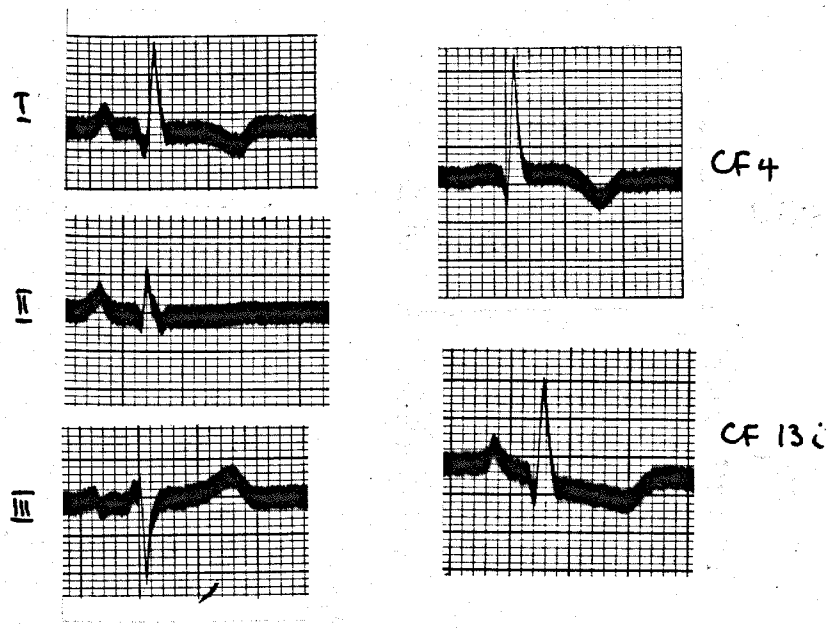


Figure 36. Case No. 3. T.McG.
electrocardiogram 12.1.1949.

In March 1949, he began to have attacks of cardiac asthma and had to give up his work. In mid-August 1949 he developed swelling of the legs and other evidence of congestive failure. He was re-admitted 14th September 1949 for suitable treatment.

On 23rd Sept. 1949 there was a further attack of cardiac pain, lasting three or four hours. An electrocardiogram was recorded on 30th Sept. 1949. It shows sinus rhythm and a long PR interval (Figure 37). There was a depressed ST_1 and elevated ST_3 with a prominent Q_3 and Q_2 was evident. The apical lead showed Q waves with an embryonic R indicating anterior infarction. CF13i showed a broad Q and a

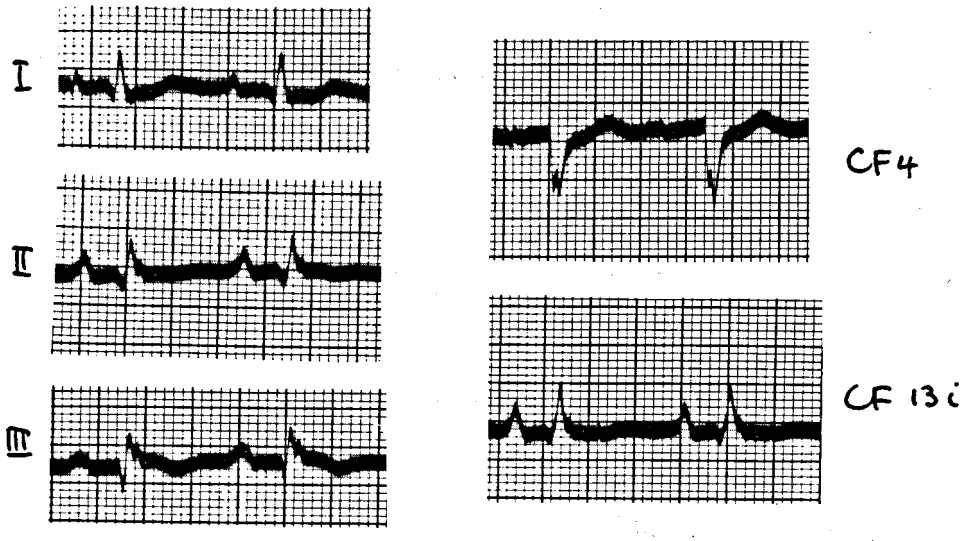


Figure 37. Case No. 3. T.McG.
Electrocardiogram 30.9.1949.

barely inverted T.

An haemoptysis occurred and the patient died that day (30.9.1949). The terminal event was thought to have been a pulmonary embolus.

Autopsy.

Pitting oedema of the legs was noted but there was no ascites or pleural effusion. The liver was enlarged and congested.

There was thrombosis in the femoral veins of both legs and many recent pulmonary emboli. The heart (weight 590 grams) showed advanced coronary artery atheroma with almost complete occlusion in the main branches of both right and left arteries.

There was a recent infarct in the posterior wall of the left ventricle and extensive mural thrombus overlying it.

The serial slices are shown in Figure 38. The first shows little of note. Sections 2 and 3 are shown enlarged in Figure 39. The posterior wall of the left ventricle and the adjacent septum show a recent yellowish infarct with a hyperaemic margin. There is an overlying ante-mortem thrombus. From the infarct patchy fibrosis extends to right and to left into the lateral and anterior walls of the left ventricle. The anterior descending branch of the left coronary artery shows gross sclerosis. Section 3 shows some endocardial thickening postero-laterally. Section 4 shows patchy fibrosis anteriorly and laterally. This is also seen in Sections 5 and 6 together with much ante-mortem and post-mortem clot. There is no noteworthy feature in the apical region.

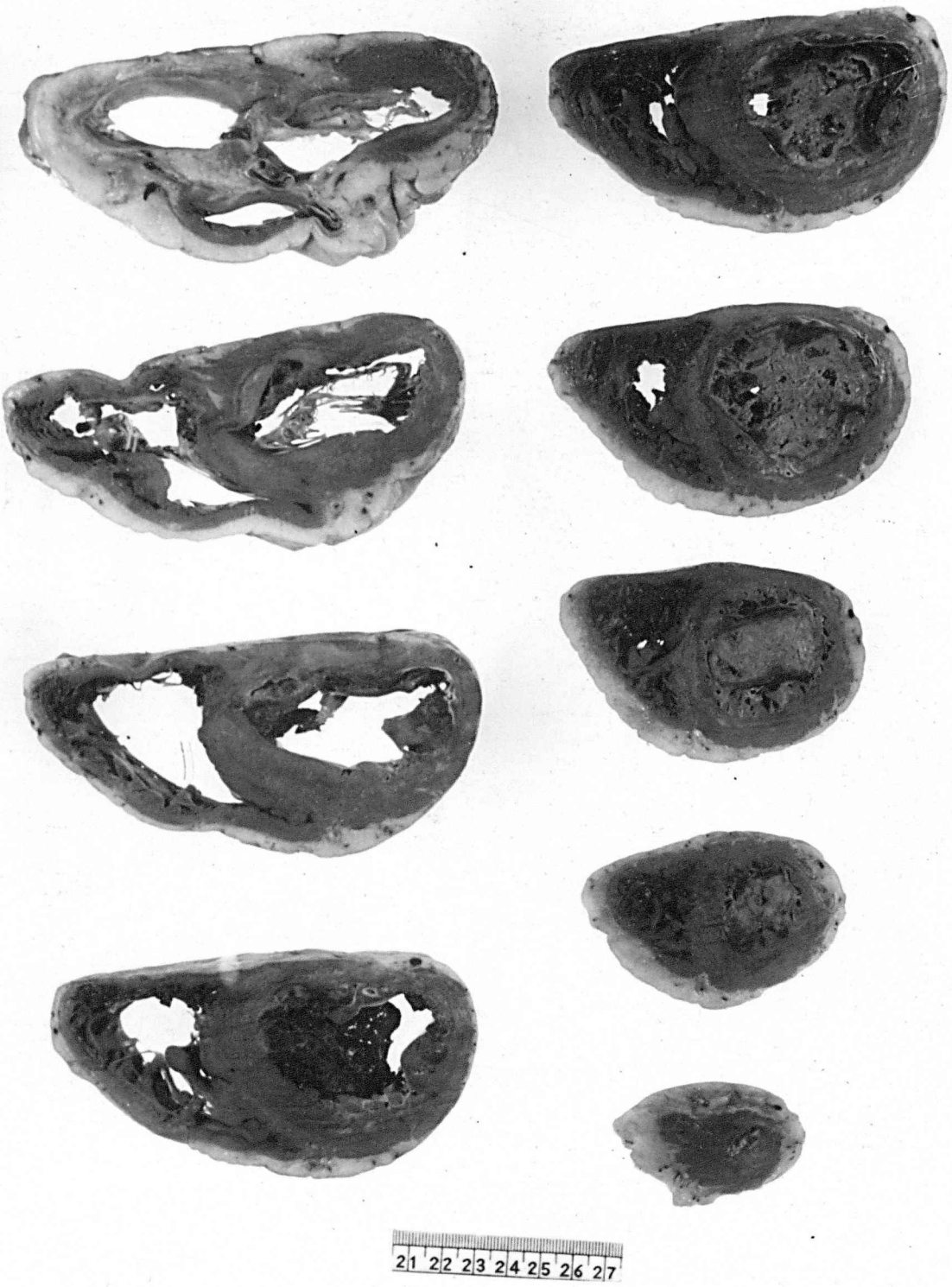


Figure 38. Case No. 3. T.McG.
Serial slices of the heart.

Figure 39. Sections 2 and 3 of Figure 38 enlarged.

Summary.

Myocardial infarcts 1944, 1948 (anterior) and 1949 (posterior). Autopsy revealed a recent posterior infarct and old antero-lateral fibrosis. The electrocardiographic records were compatible with these findings. The clinical age of the recent infarct (7 days) agrees with the criteria of Mallory et al. (1939).

Comment.

The final electrocardiographic pattern might have been accounted for by acute pulmonary embolism; but the Q wave changes suggested a posterior infarct which was confirmed.

Case No. 4.

P.D., a labourer aged 63, was admitted to hospital from the Casualty Department on 20th February 1949. He was collapsed and no detailed history was obtainable, but he had had pain in the chest for about eight hours. He was cyanosed and dyspnoeic. Blood pressure 130/80. He became seriously ill and died after ten days.

A standard lead electrocardiogram was taken 21st February 1949 and showed the picture of an anterior infarct (Figure 40).

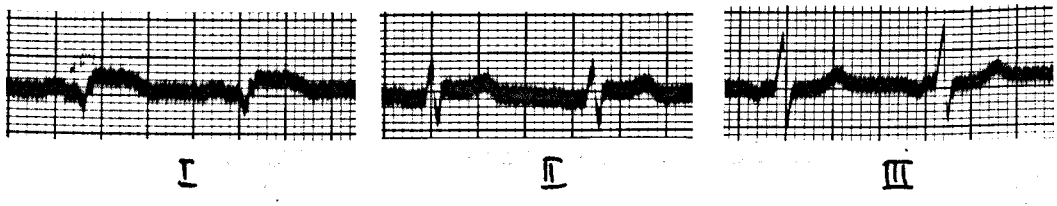


Figure 40. Case No. 4. P.D.
Electrocardiogram 21.2.1949

A second record was made on 1.3.1949 and showed little change, but chest leads were included. (Figure 41).

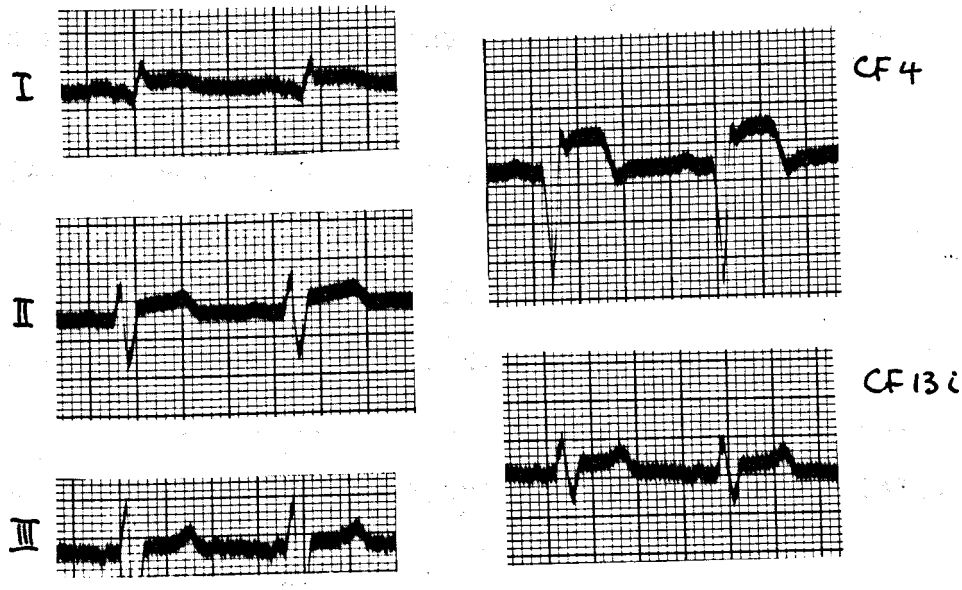


Figure 41. Case No. 4. P.D.
Electrocardiogram 1.3.1949

CF4 shows the characteristic picture of a recent anterior infarct. It is noteworthy that CF13i shows ST elevation, suggestive that the anterior infarct extends laterally to the left lateral wall.

Autopsy.

An obese man. Bilateral pleural effusions were present; there were a few emphysematous bullae

in the lungs but no congestion. The remaining viscera, apart from the heart, were normal.

The heart (weight 510 grams) showed several fibrinous pericardial adhesions. An infarct was seen involving the apex and the anterior and lateral walls of the left ventricle. A thrombus occluded completely the anterior descending branch of the left coronary artery, 2 cms. from its origin. There was very little aortic atheroma. The heart is seen in Figure 42 A and B.

The serial slices are shown in Figure 43. It will be noted that the arteries stand out plainly. They were injected with a barium sulphate - sodium alginate injection mass. The radiographs were indifferent and are not shown.

Section 1 shows gelatinous looking areas in the anterior half of the septum and in the anterior wall. Section 2 shows a large yellow infarct with gelatinous margins situated anteriorly and in the anterior part of the septum with overlying ante-mortem clot. Section 3 (and Section 6) are shown enlarged in Figure 44. The infarct is more extensive as one proceeds downwards. Section 6 shows the left ventricle almost encircled by infarct and in Section 7 it is

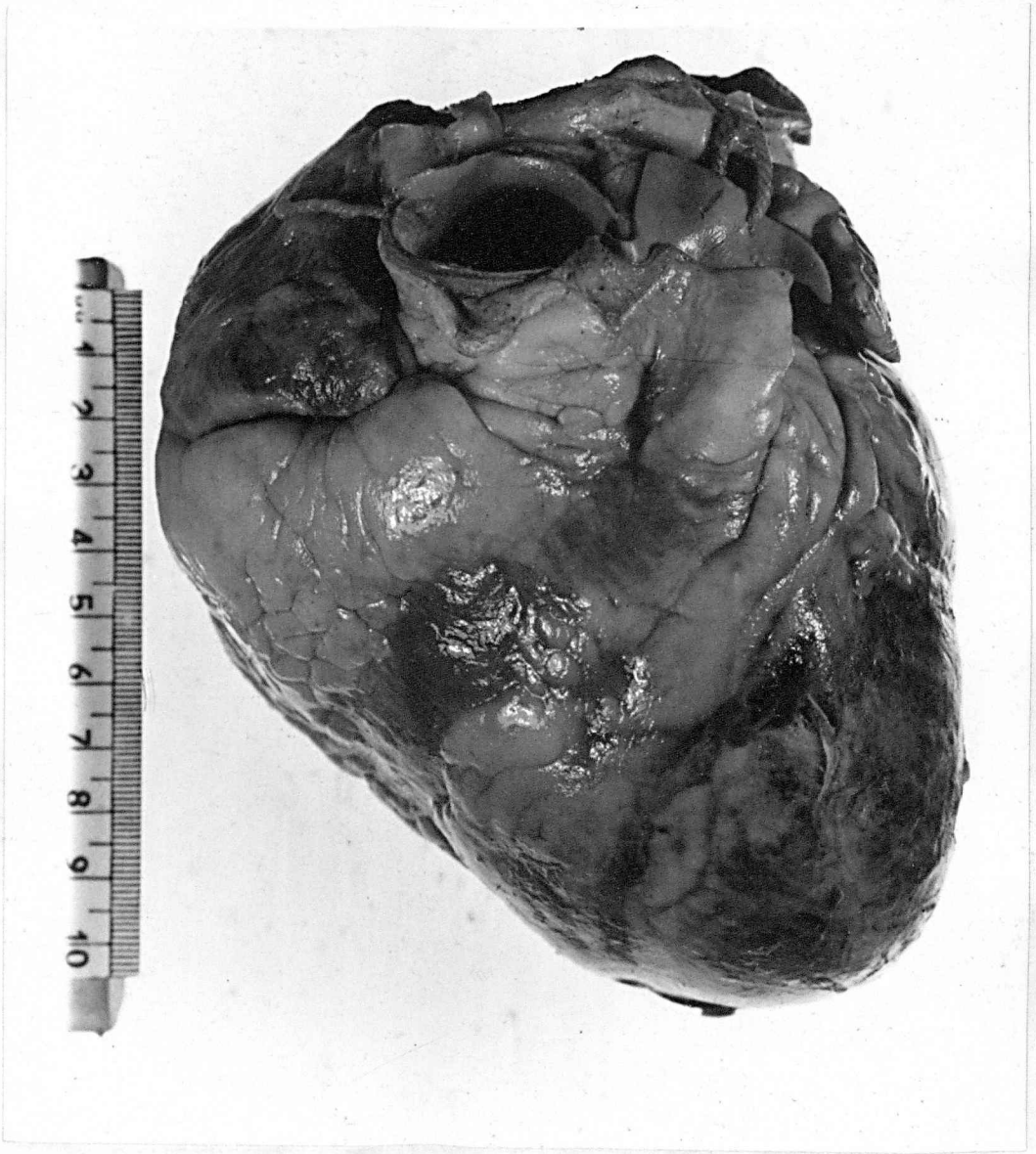


Figure 42 A. Case No. 4. P.D.
Anterior aspect of heart.

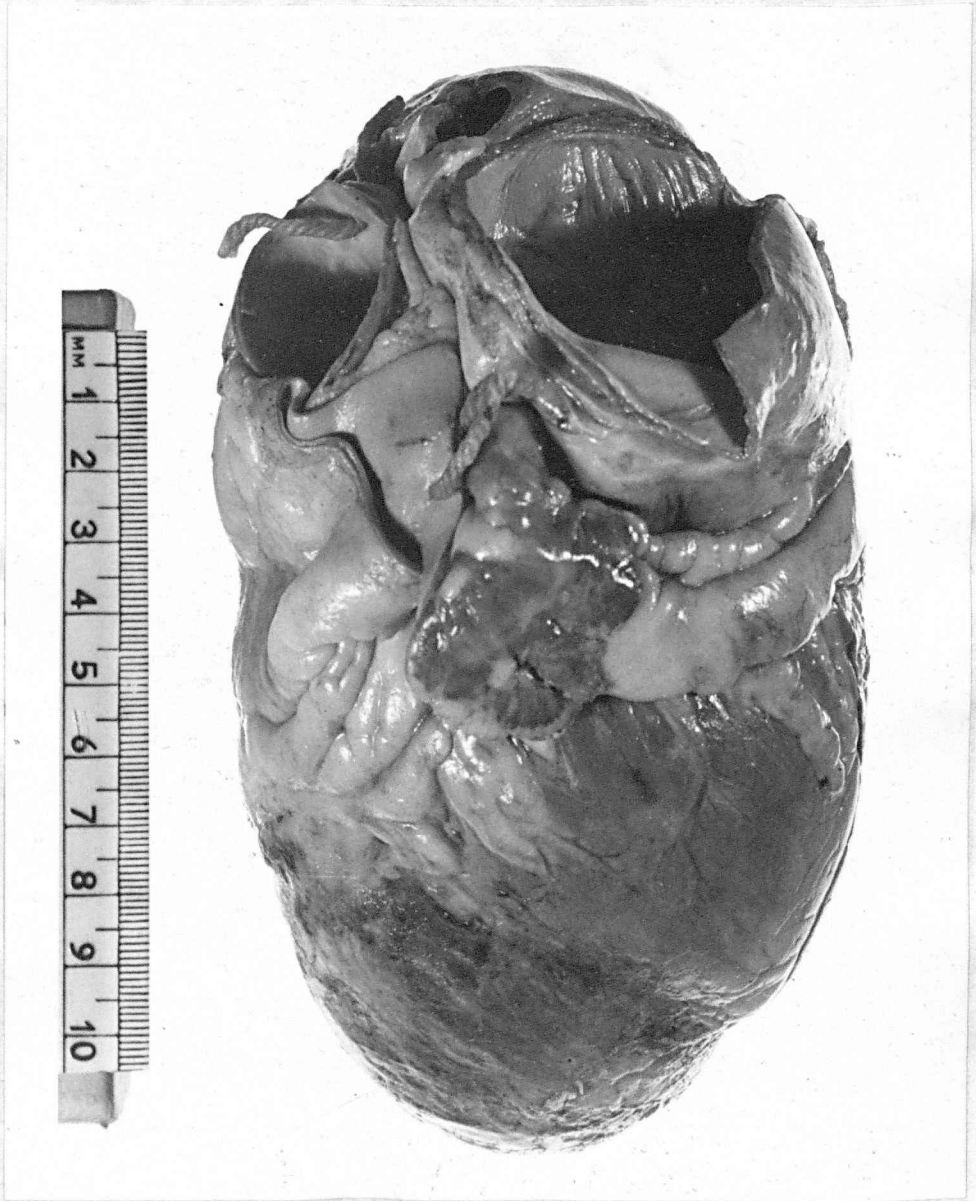


Figure 42 B. Case No. 4. P.D.
Left lateral aspect.

Figure 43. Case No. 4. P.D.
Serial slices of the heart.

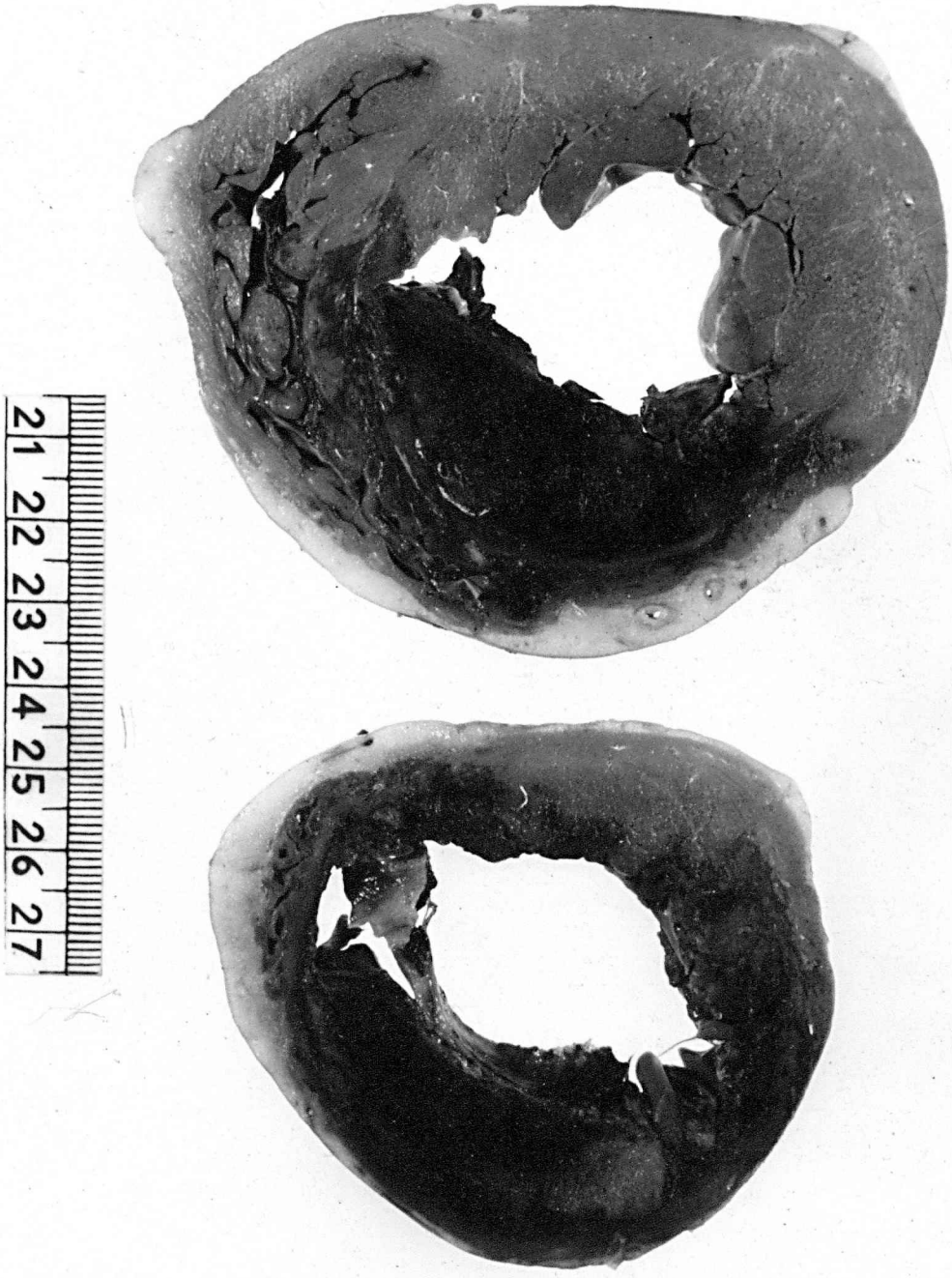


Figure 44. Case No. 4. P.D.
Sections 3 and 6 of Figure 43.

so encircled. Relatively healthy looking muscle can only be seen at the postero-lateral angle where it fringes the infarct. Sections 8 and 9 show infarct and ante-mortem thrombus.

Summary.

This case appears to have been one of a first, overwhelming myocardial infarct. It was about 10 days old, and the macroscopic appearances are in accordance.

Comment.

The shape of the infarct is such that towards the base of the heart it is antero-septal. Towards the apex it encircles the heart and may be compared to a cup with a tongue projecting upwards anteriorly.

The electrocardiograms denote a predominantly anterior infarct, but the change in CF13i denoted that the infarct extends backwards in the lateral wall somewhat.

Case No. 5.

W.P., a 70 year old retired electrician, was referred to an Out-patient Clinic on 19th November 1948 because of vertigo and strangeness in his behaviour. There were no previous illnesses and he denied venereal infection. Two months previously he had noted giddiness and a slight cough. He had also some difficulty in getting his breath. (The history was not entirely satisfactory.)

On examination he was apathetic. There was auricular fibrillation (120 per minute), the systolic blood pressure was about 140 and there was a systolic murmur at the apex. The chest showed diminished movement and absent breath sounds at the left base. There was ankle swelling and albuminuria. The patient was admitted, and digitalisation commenced, with effective control of the ventricular rate.

An electrocardiogram was taken the following day. It confirmed the diagnosis of auricular fibrillation and showed right axis deviation with low voltage. Q_1 and Q_2 waves were shown. The QRS interval was full. CF4 showed deep Q5 waves. T in CF13i was flat. (Figure 45). The record was compatible with an anterior infarct.

Figure 45. Case No. 5. W.P.
Electrocardiogram 20.11.1948

The blood and cerebrospinal fluid Wassermann reactions were negative and the cerebrospinal fluid showed: protein 25 mgm.%, 1 lymphocyte per cu.mm.

An x-ray of the chest showed obliteration of both costo-phrenic angles, particularly the left, and considerable cardiac enlargement, mainly of the left

ventricle. There was moderate general pulmonary fibrosis. (The film was not available for reproduction.)

Sixteen days after admission, there was a sudden pain in the right leg and gangrene developed. A right lumbar sympathetic block was performed and heparin administration begun (6.12.48). It became evident, however, that amputation would be necessary and a high thigh amputation was carried out on 21st December 1948. After four days the patient died.

Autopsy.

There were pleural adhesions. The left kidney showed old and recent infarcts, and in the right occipital lobe of the brain a large recent area of softening. The cerebral arteries were atheromatous. There was also an older smaller area of softening in the right internal capsule. There were recent infarcts in the spleen. The right common iliac artery was blocked by thrombus which extended down the femoral arteries to the amputation stump.

The heart weighed 505 grams. There was thrombus in the left auricular appendage. The front of the left ventricle showed a patch of calcification and there were light pericardial adhesions overlying it.

The aortic valve cusps were thickened and partly adherent. The aorta was atheromatous.

The external appearance of the heart is shown in Figures 46 and 47.

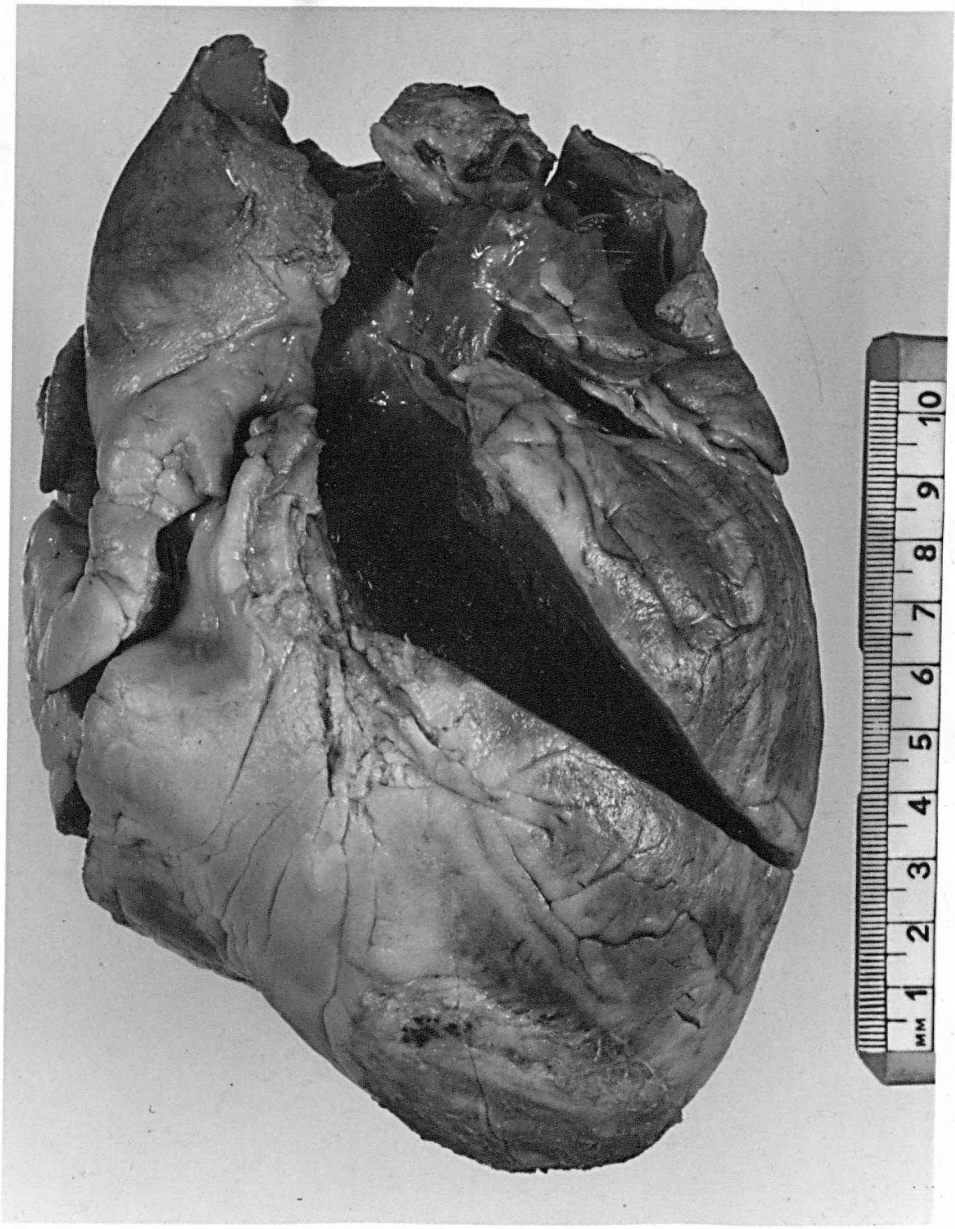


Figure 46. Case No. 5. W.P. Anterior view of the heart.

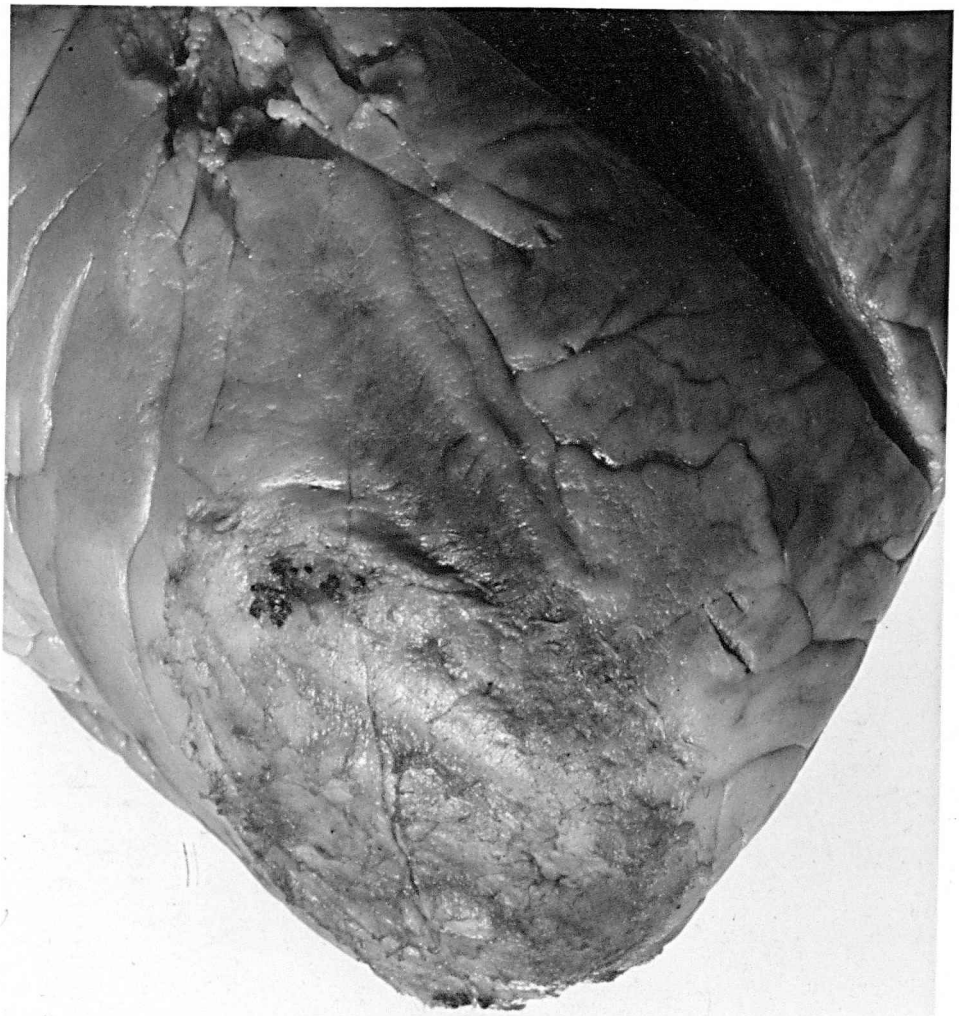


Figure 47. Enlarged view of the apex.

In view of the fact that the infarct appeared to be calcified, x-ray pictures were taken of the heart, and these are shown in Figures 48 and 49.

Figure 48. Antero-posterior radiograph of the heart. Note the calcified coronary arteries.

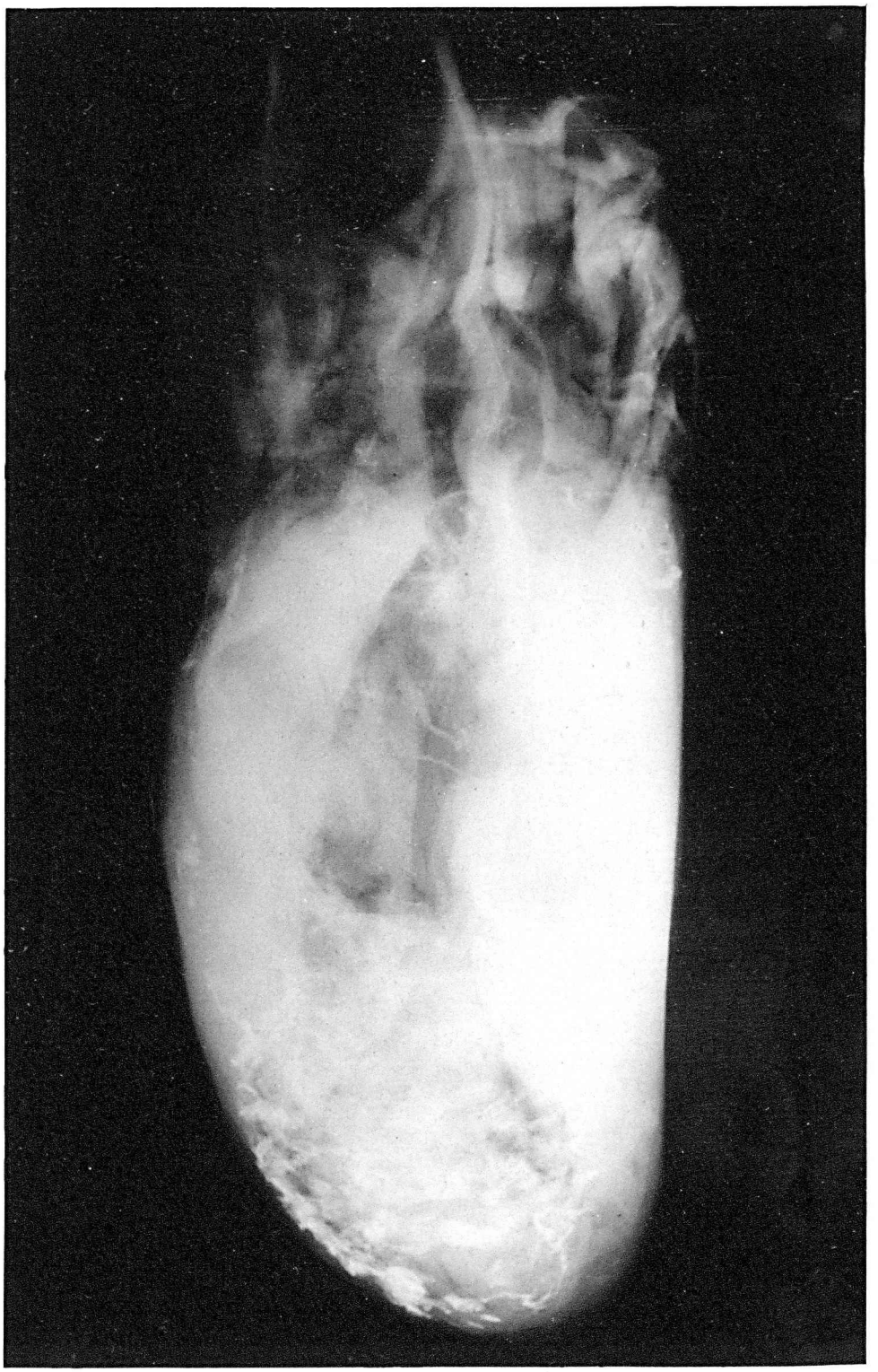


Figure 49. Left lateral radiograph.

The heart was cut into serial slices, which are shown in Figure 50. Section 1 showed fibrosis in the lateral wall of the left ventricle. There are reddish areas in the septum and posteriorly. Section 2 shows patchy fibrosis anteriorly and laterally and reddish patchy areas posteriorly. In section 3 there is patchy fibrosis laterally and more dense fibrosis anteriorly, and extending half-way into the septum. The endocardium in this site is thickened and in the naked eye specimen calcification could be seen there also. Section 4 shows fibrosis in the septum and present in a patchy distribution anteriorly and laterally. The cavity of the left ventricle contains a chicken fat clot. Section 5 shows extensive septal fibrosis and calcification with thinning and reddish areas situated laterally (Figure 51). In section 6 there is a dense patch of fibrosis at the posterior border of the septum running into the posterior walls of the right and left ventricles; chicken fat clot in the left ventricular cavity. Section 7 shows much dense fibrosis almost encircling the ventricle.

Figure 50. Case No. 5. W.P. Serial slices of the heart.

Figure 51. Sections 4 and 5 of Figure 50 enlarged.

Owing to the presence of calcification in the infarct, a radiograph of the serial slices is shown. The extent and situation of the infarct is clearly defined. Calcification in the coronary arteries is likewise indicated.

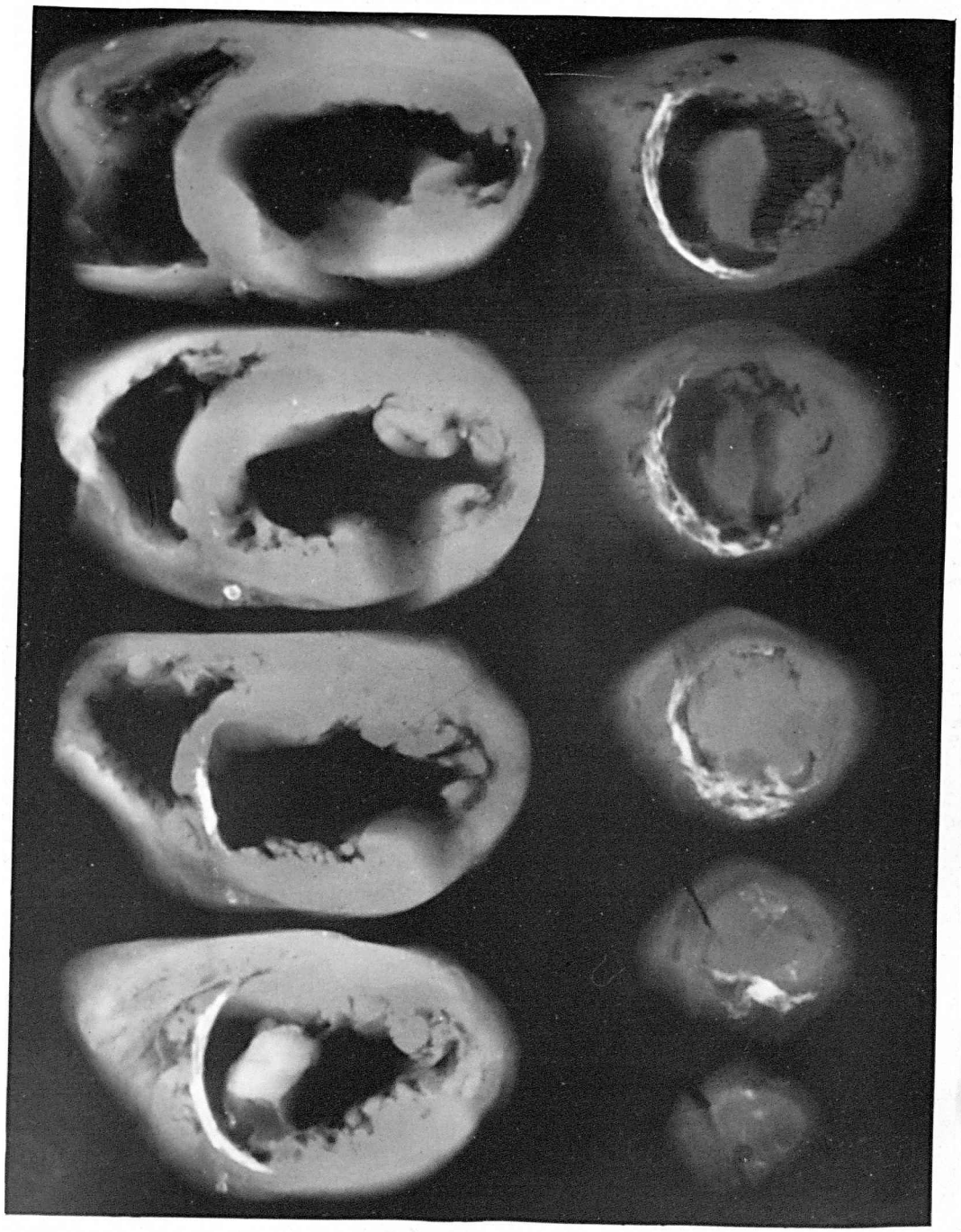


Figure 52. Radiograph of the serial slices to show the calcified infarct. For comparison with Fig.50.

Summary.

There was no history obtainable of myocardial infarction in this case, which was one of auricular fibrillation. Although systemic emboli had probably occurred previously, digitalisation appears to have precipitated more.

Comment.

The electrocardiogram showed evidence of an old anterior infarct by the QS deflections in the apical lead. An old calcified antero-septal infarct was found at autopsy with probable recent areas of infarction posteriorly. The value of the standard leads and CF13i is reduced owing to the irregular contractions resulting from auricular fibrillation. CR1 is shown as it is a useful lead for showing this arrhythmia.

In its clinical and pathological features this case closely resembles one reported by Hirschboeck (1934).

Case No. 6.

S.W., an analytical chemist aged 54, was first seen in the Heart Clinic in March 1947. One month previously he had had an attack of substernal pain radiating across the front of the chest, which lasted three days and was associated with dyspnoea. He was kept at rest in bed for three weeks.

There had been no significant previous illnesses. The patient was of sthenic build and high colour. The heart was not enlarged clinically. There was a soft systolic murmur in the aortic area; the blood pressure was 165/90. The lung bases were clear and the urine normal.

The electrocardiogram (Figure 53A) showed evidence of a posterior infarct in the standard leads, there being ST segment elevation in leads II and III. After a further period of rest at home he was seen again on 15th April 1947 and the electrocardiogram is shown (Figure 53B). He was experiencing some pain and tightness in the upper chest on walking. Blood pressure 175/100.

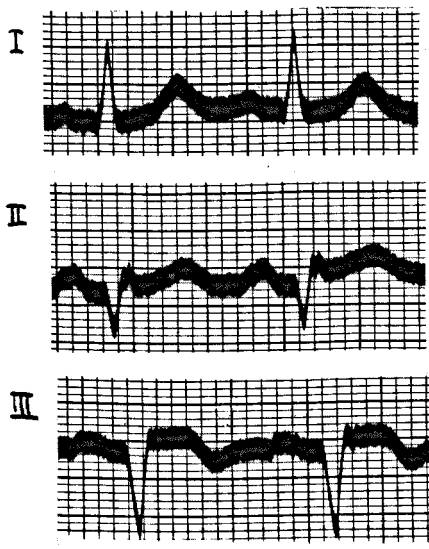


Figure 53A. 26.3.47

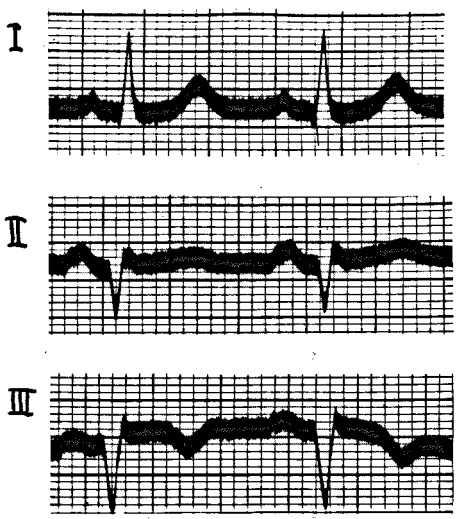


Figure 53B. 15.4.47

The patient was considered fit to go away for convalescent treatment and he was seen again in May 1947. He had developed a pneumonic infection when away, and x-ray examination showed a resolving pneumonic infiltration in the right middle lobe. The cardiac outline was within normal limits. The patient resumed work.

He was next seen in November 1947, and there was still some evidence of local fibrosis in the right lung. The electrocardiogram was unchanged.

In June 1948 the chest x-ray showed moderate general pulmonary fibrosis but the lung fields were otherwise clear (Figure 54).

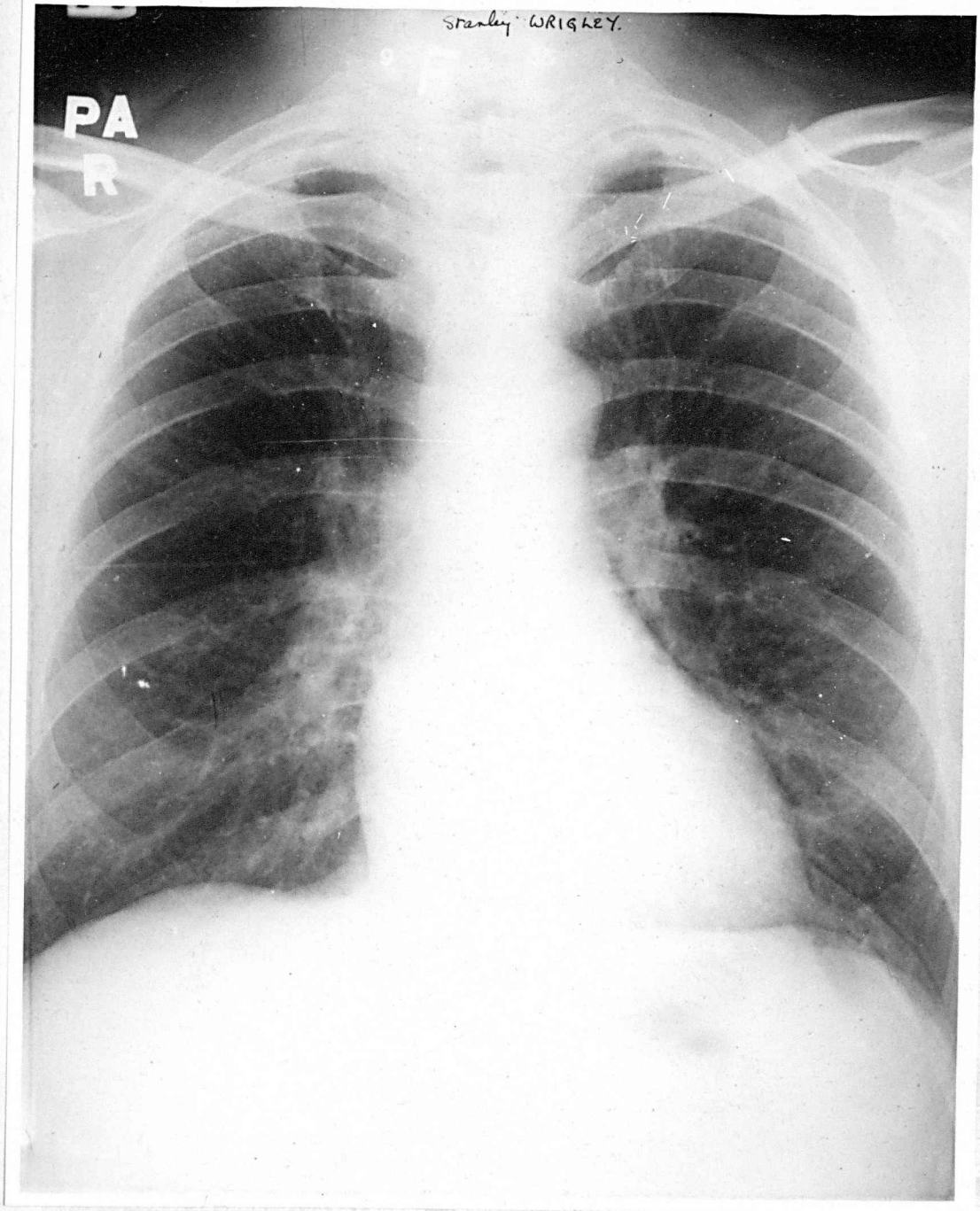


Figure 54. X-ray of chest, 9.6.48.

The electrocardiogram showed a deep Q_2 and Q_3 with a negative T_3 (Figure 55).

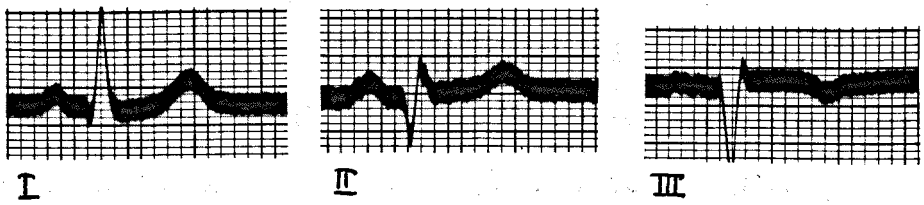


Figure 55. Electrocardiogram, 9.6.48.

He attended in April 1949 because he had had two syncopal attacks. The electrocardiogram had changed so that T_2 was now inverted slightly; and the T in CF13i showed this change more markedly (Figure 56).

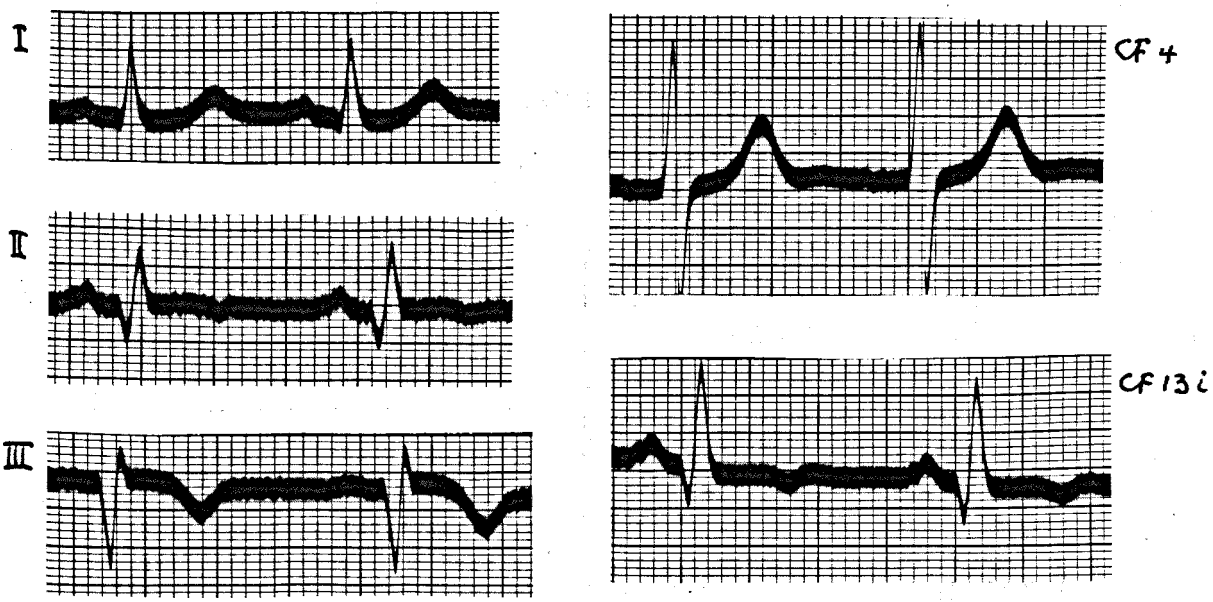


Figure 56. Electrocardiogram, 13.4.49.

In August 1949 he began to suffer from intermittent claudication in the left leg. In March 1950 he complained of flatulence and fluttering sensations in the chest, but he had business worries and anxiety regarding his health, so that some of his symptoms seemed to be of psychogenic origin.

He was seen once more on 5th July 1950. He had newly returned from a holiday when he was walking three miles slowly but without angina or claudication. After his return, however, the angina recurred and a few days before attending the hospital had had to stop four times in a distance of a quarter of a mile. Examination showed nothing of note; the blood pressure was 210/120.

Another electrocardiogram was taken on 12th July, and showed that T_2 and T in CF13i were both upright (Figure 57).

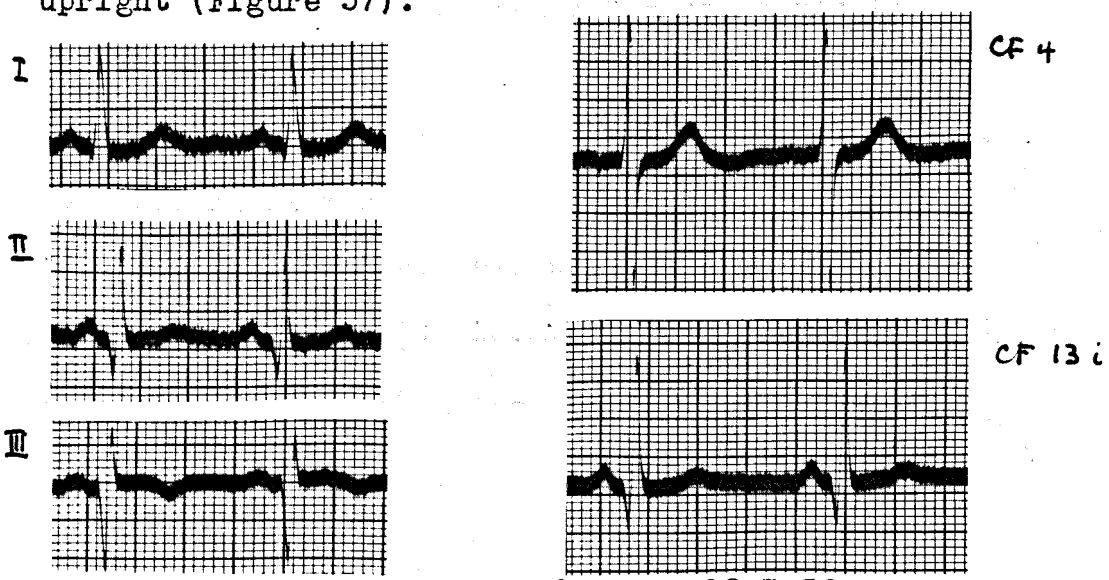


Figure 57. Electrocardiogram, 12.7.50.

On 15th July there was another severe attack of pain in the chest but the duration must have been short for the patient went to work the following day. He was brought home because the pain recurred; and it persisted for seven or eight hours. Two days later (18.7.50) the patient was admitted to hospital with more pain. He was dyspnoeic, cyanosed and collapsed. He died three hours after admission. No further electrocardiogram had been recorded since 12.7.50.

Autopsy.

There was a small amount of fluid in each pleural cavity, but not in the abdomen. The lungs were slightly congested but showed no sign of the old pneumonia. The aorta showed a moderate degree of atheroma and this was present in the basilar artery also. The kidneys showed a few depressed sub-capsular scars.

The heart weighed 510 grams. There were no pericardial adhesions. There was scarring at the apex of the left ventricle especially posteriorly. There was an extensive infarct affecting the anterior lateral and posterior walls of the left ventricle and

mural thrombus at the apex. The anterior descending branch of the left coronary artery was recently occluded, and both coronary arteries were extensively atheromatous.

The serial slices of the heart are shown in Figure 58.

Section 1 shows a pale area in the anterior wall of the left ventricle indicative of a recent infarct. The descending branch of the left coronary artery may be seen occluded. Section 2 shows the same anterior infarct and much fibrosis in the posterior wall. In this and subsequent sections the right ventricle shows chicken fat clot. The appearances in sections 3 and 4 are similar. Ante-mortem thrombus may be seen in the left ventricle. Sections 5 and 8 are seen enlarged in Figure 59. The later sections show fibrosis encircling the left ventricle.

Summary.

This patient had a posterior infarct three years before death. In the interval he probably had a number of minor thrombotic episodes which were terminated by an anterior infarct from which he died three days later. All the electrocardiographic evidence

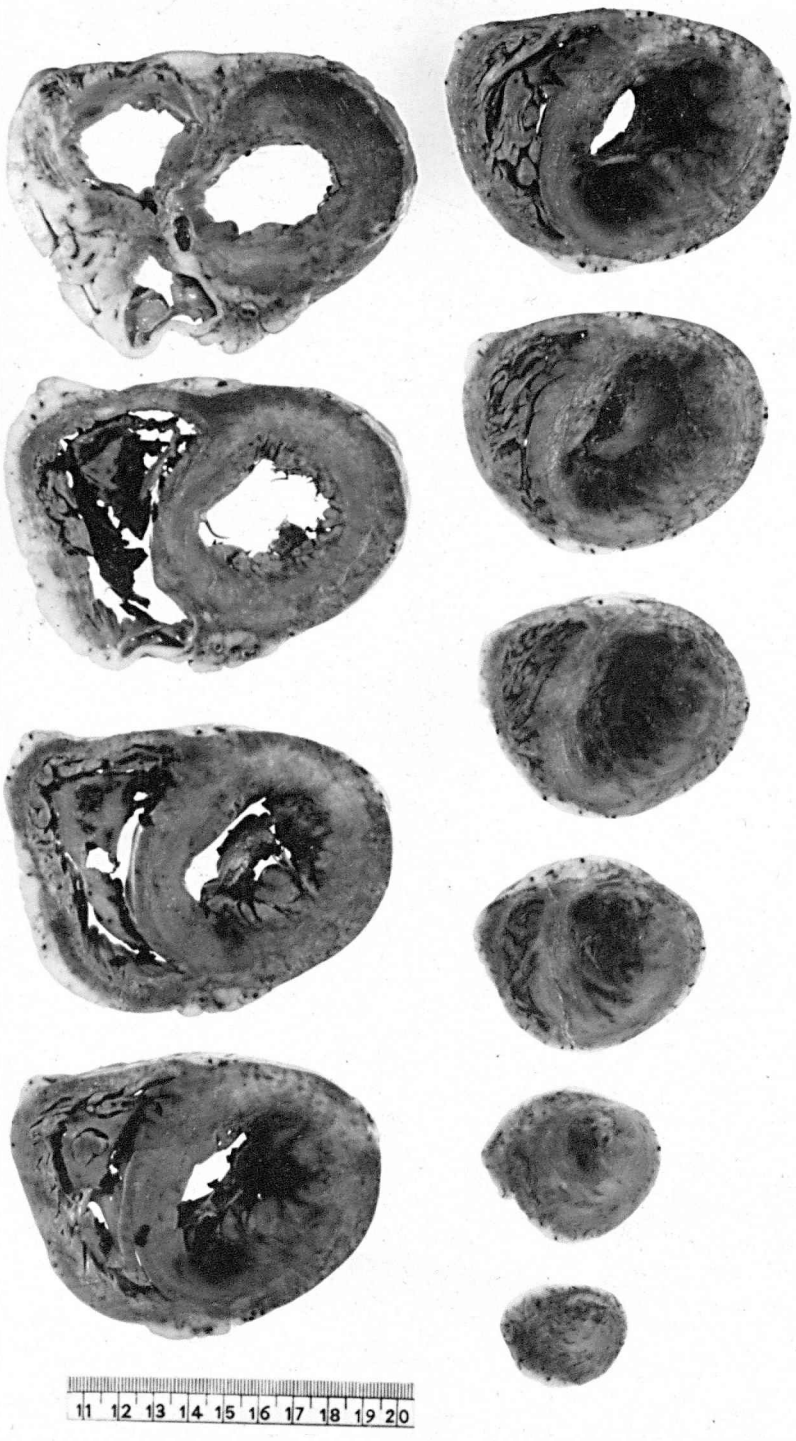


Figure 58. Case No. 6. S.W. Serial slices of the heart.

Figure 59. Sections 5 and 8 of Figure 58, enlarged.

was collected before the final incident and shows posterior infarction.

Comment.

Beyond indicating which is old and which is new, one can say little regarding the ages of the infarcts. The appearance of the recent anterior one is compatible with an age of three days. The fibrosis probably resulted from several coronary artery occlusions.

Case No. 7.

A.B., a retired publican aged 72, was seen in the Heart Clinic 12th July 1950. He had had effort angina for 5 or 6 years and had been known to be hypertensive for ten. Apart from a reputed inflammation of the kidneys at the age of 27 necessitating two weeks in bed, he had always been healthy. One son had died of tuberculosis.

Physical examination showed a well preserved looking man. There was slight cardiac enlargement but no evidence of any valvular lesion. The blood pressure was 225/120. The urine was normal. An x-ray of the chest that day (Figure 60) was reported: "Scattered opacities in both infraclavicular regions. The appearances are those of a tuberculous lesion of doubtful activity. Normal cardiac outlines with a little unfolding of the aorta."

On the day of his attendance, he had awakened with pain in the chest. The electrocardiogram showed slight ST sagging in lead I and a flat T in CF13i (Figure 61).

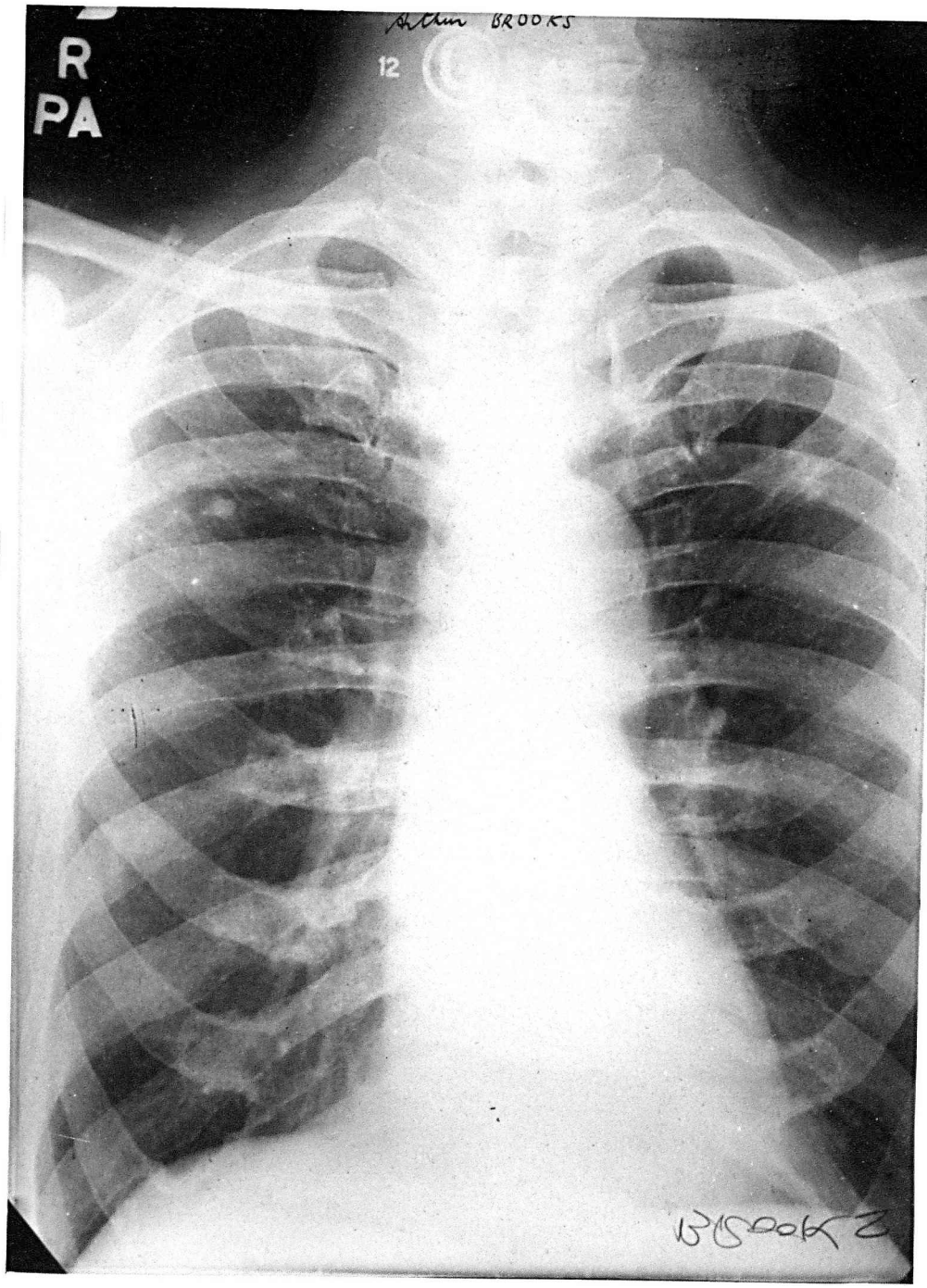


Figure 60. Case No. 7. A.B. Chest x-ray.

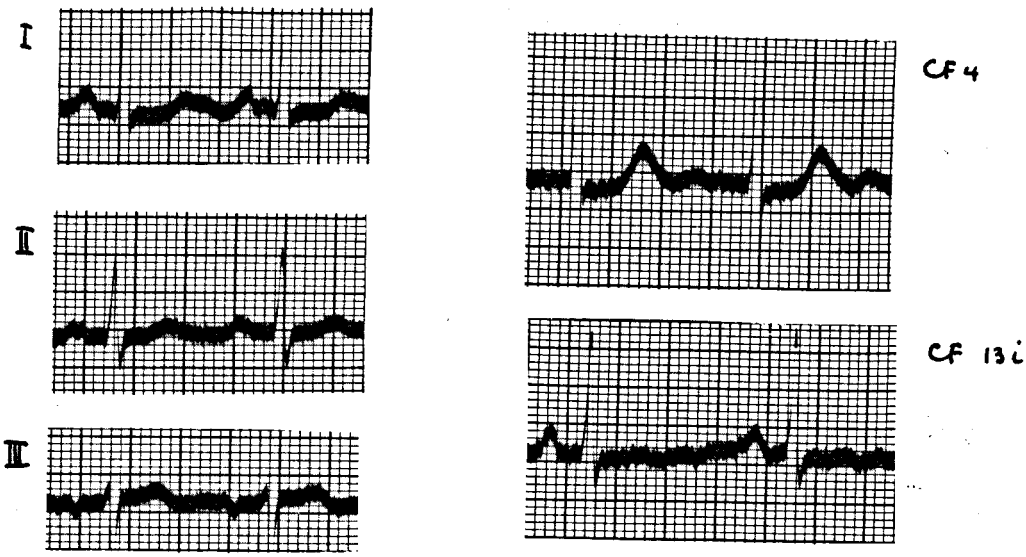
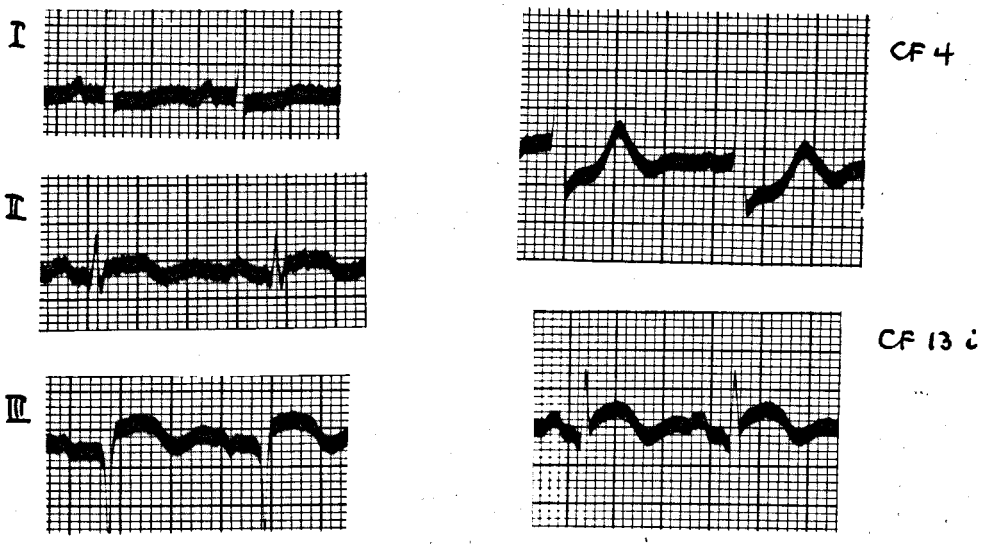


Figure 61. Case No. 7. A.B. Electrogardiogram 12.7.1950

Four days later, he awakened with chest pain of considerable severity. It continued throughout the day and on the day following he was admitted (17.7.1950).

On admission he was in obvious pain, was restless and slightly cyanosed. The pulse was regular but interrupted by premature beats. There was an apical systolic murmur and the second aortic sound was accentuated. Blood pressure 170/120. The chest showed no abnormality. In hospital, the pains recurred and required morphine frequently. On the day after admission the blood pressure was 145/100 and three days later 130/100, when his condition was deteriorating.

Electrocardiograms were taken on 19th and 21st July and are shown in Figure 62 A and B.



Figu 62A. Case No. 7. A.B. Electrocardiogram 19.7.1950

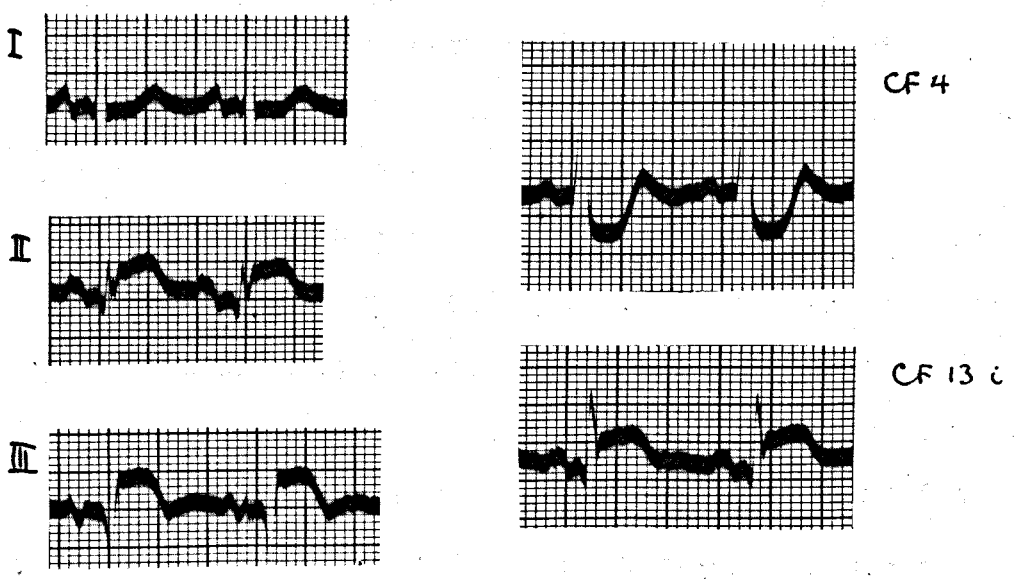


Figure 62B. Case No. 7. A.B. Electrocardiogram 21.7.1950.

Later in the day of 21st July a friction rub could be heard medial to the apex and the blood pressure fell to 110/70. Cheyne-Stokes breathing followed and two days later (23.7.1950) the patient died.

The blood Wassermann reaction had been found to be negative.

Autopsy.

The body was that of a well built old man. There was a small amount of clear yellow fluid in the left pleural cavity. The lungs showed a few peripheral bullae in the upper lobes. The parenchyma was congested but not oedematous. The kidneys were both severely and irregularly scarred with some granularity of the surface between the scars. The cortex was irregularly thinned. The renal arteries and the circle of Willis showed gross atheroma.

The heart weighed 630 grams. The parietal pericardium was lightly adherent anteriorly and posteriorly. The left lateral and posterior surfaces of the heart showed recent fibrinous exudate and a markedly haemorrhagic appearance. Both coronary arteries were occluded apparently by atheroma (Figures 63 and 64).

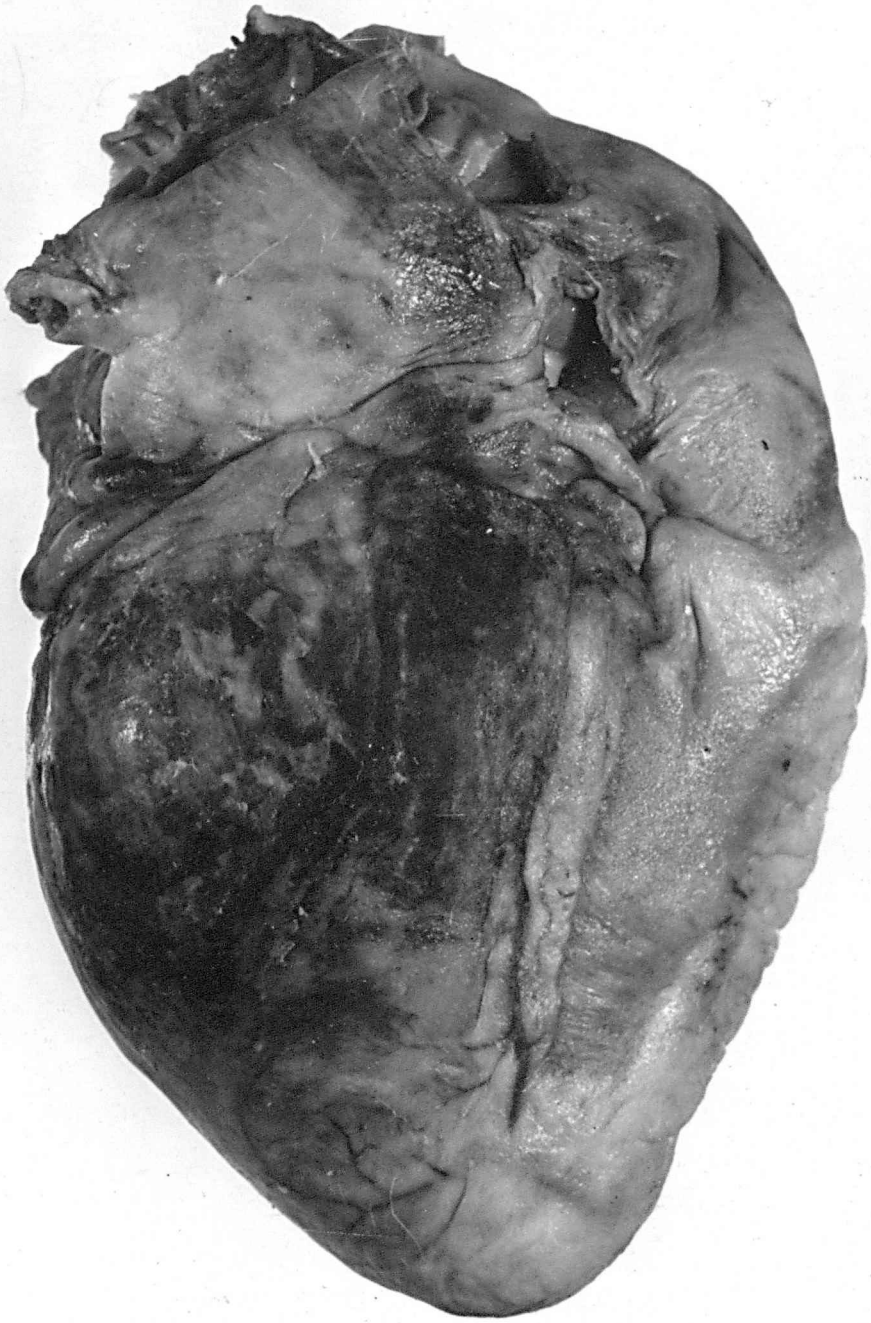


Figure 63. Case No. 7. A.B. Posterior surface of the heart.

Figure 64. Case No. 7. A.B. Left lateral surface of the heart.

It may be noted that the left auricular appendage shows the haemorrhagic appearance of the ventricle; and that one of the electrocardiograms shows auricular premature beats (Figure 62A).

The serial slices are shown in Figure 65. There is considerable concentric hypertrophy of the left ventricle, compatible with long standing hypertension. Section 1 shows a large postero-lateral infarct and section 2 shows it running into the posterior aspect of the septum. The haemorrhagic appearance of the overlying epicardial surface will be noted. Sections 3 and 5 are shown enlarged in Figure 66. Section 3 shows the infarct to be postero-lateral and the papillary muscles are involved. Mural thrombus may be seen in this and succeeding sections. In Section 6 there are a few gelatinous looking areas in the infarct and in this and the sections following the infarct is seen to encircle the left ventricle.

Summary.

A case of long standing hypertension and slowly progressive myocardial ischaemia terminating in myocardial infarction. Some of the fibrous tissue may be replacement fibrosis. There is a very large postero-lateral infarct, and the left auricle is infarcted also.

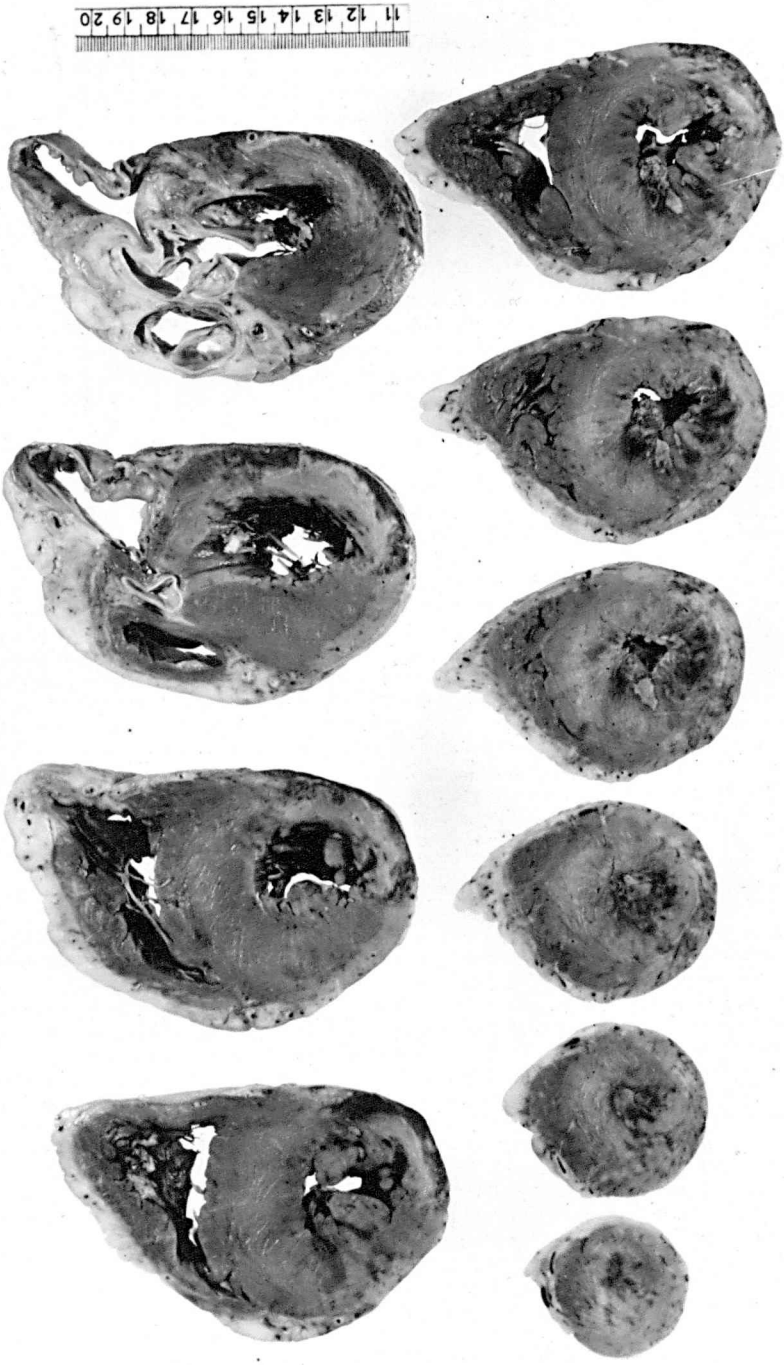


Figure 65. Case No. 7. A.B. Serial slices of the heart.

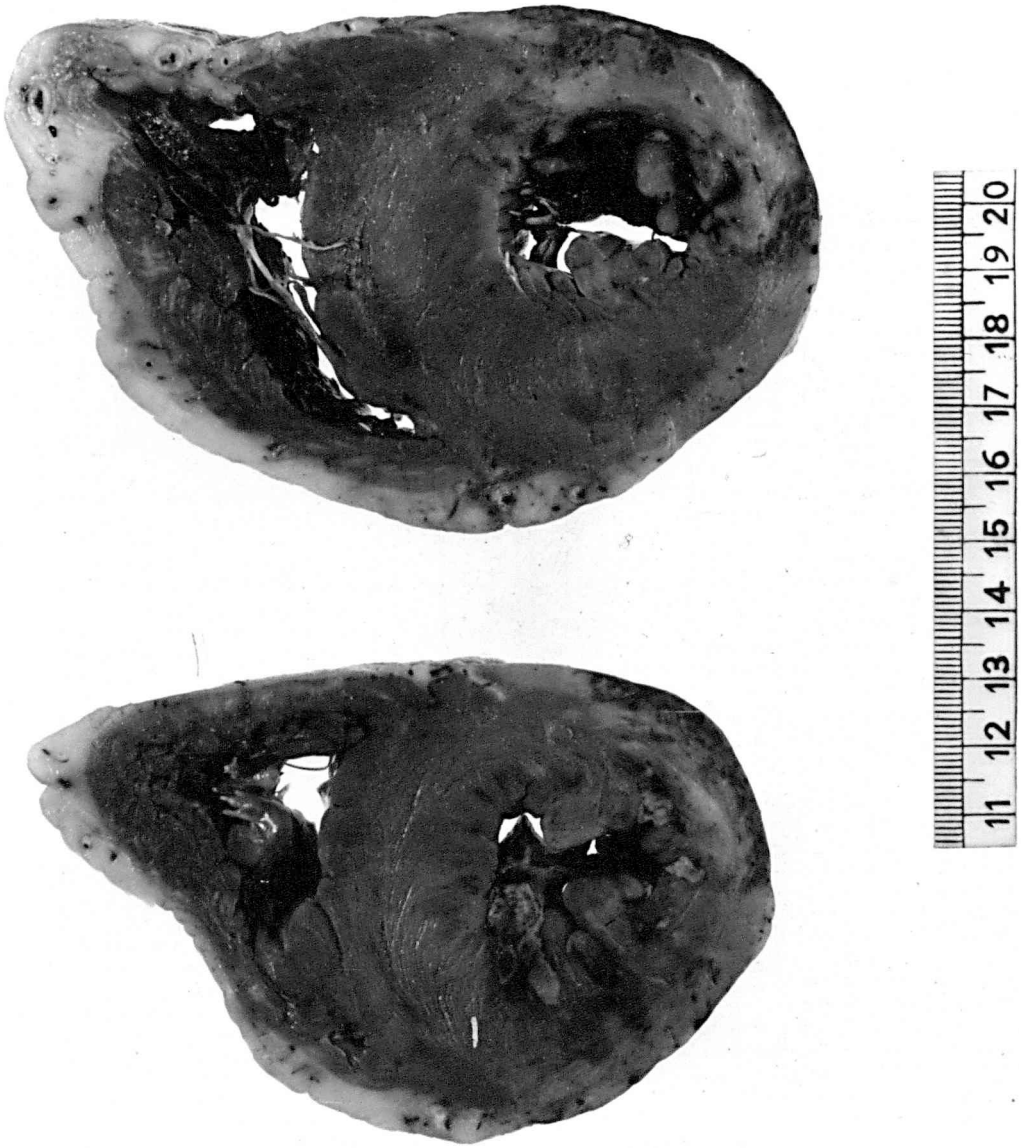


Figure 66. Case No. 7. A.B. Sections 3 and 5 of Figure 65 enlarged.

The age of the infarct is 7 days counting from the first onset of severe pain or 11 days counting from the "herald" attack.

Comment.

There is close agreement between the electrocardiographic localisation and the autopsy, but the infarct is a very large one and could scarcely fail to give a clear record. The value of CF13i is reduced since CF4 shows ST depression suggestive of the localisation, nevertheless CF13i shows the changes anticipated.

Case No. 8.

P.W., a man aged 70, was admitted as an emergency. For two years he had been suffering from angina of effort and recently attacks had been occurring at rest. He had complained of breathlessness on exertion and latterly, nocturnally also.

His father had died of a "stroke"; one brother died of a "heart attack". The patient himself had suffered from winter bronchitis for some years.

At 10.0 p.m. the day before admission an anginal pain occurred similar to those to which he was accustomed, but much worse and more prolonged. He was breathless and shocked. The pain was subsiding when he was admitted twelve hours later. There was tachycardia (100 per minute) and the heart sounds were distant. Blood pressure 135/115. Crepitations were heard all over the chest.

By 4.30 p.m. (6th November 1950) his condition was much worse and the systolic blood pressure had fallen to 65. An electrocardiogram (Figure 67) showed right bundle branch block with ST elevation in

lead I and depression in lead III. CF4 showed a deep Q with ST segment elevation and early T wave inversion. CF13i showed evidence of the bundle block but no evidence of infarction, hence it was deduced that the infarct was anterior.

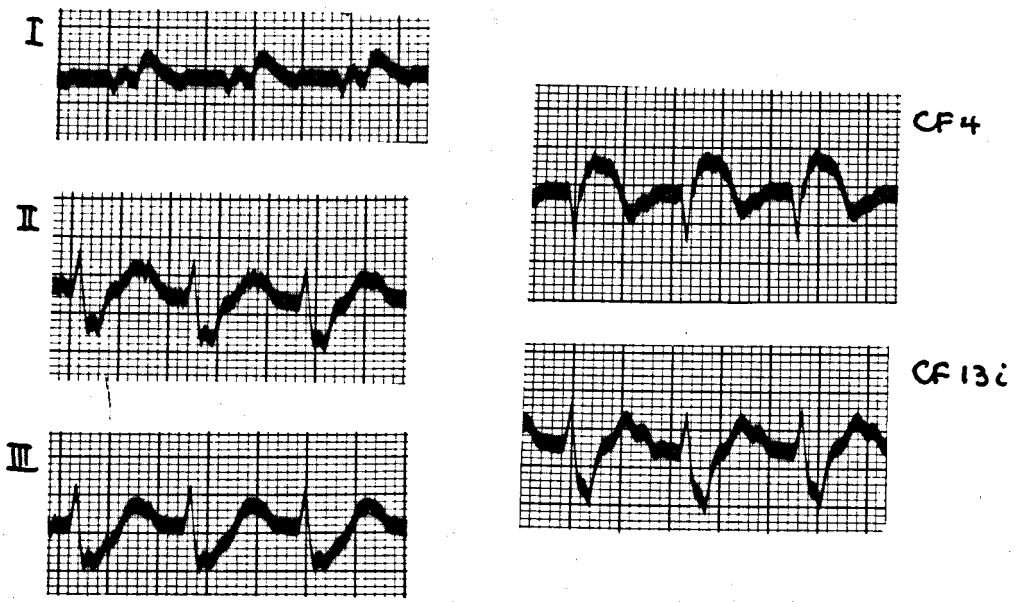


Figure 67. Case No. 8. P.W. Electrocardiogram 6.11.1950.

About 24 hours after admission the patient died.

Autopsy.

There was a small quantity of clear fluid in each pleural cavity but none in the abdomen. The lungs showed marked diffuse emphysema of both lower lobes and anterior margins of the upper. Posteriorly, both lungs were oedematous and the bronchi contained muco-pus.

The aorta showed a mild degree of atheroma in its thoracic part; in the abdomen it showed calcified plaques some of which had ulcerated and showed ante-mortem thrombus.

The heart weighed 530 grams. There were no pericardial adhesions, but there was a small amount of turbid fluid in the pericordial sac. There were petechial haemorrhages over the apex of the left ventricle and in the region of the auriculo-ventricular sulcus on the left side and milk spots over the right ventricle anteriorly and posteriorly. The coronary arteries were atheromatous.

The serial slices in this case have been spoilt to some extent by irregular fixation. Nevertheless they show the infarct (Figure 68) fairly clearly.

Figure 68. Case No. 8. P.W. Serial slices of heart.

Section 1 shows a yellowish infarct situated exactly in the anterior wall of the left ventricle, and this is also seen in Section 2. Both ventricles contain post-mortem clot, but that in the left ventricular cavity in Section 1 was loose and was discarded. Sections 3 and 6 are shown enlarged in Figure 69. Section 3 shows the upper limit of an ante-mortem clot attached over the surface of the infarct and this is seen better in Section 4, where the infarct is seen to extend somewhat laterally (to the left). The admixture of haemorrhagic areas in the infarct may be seen in this and in Section 5. In Section 6 the infarct is extending into the septum; and in Section 7 it all but encircles the left ventricle, the posterior part of the septum being relatively normal. Section 8, the apex, shows irregular yellow areas.

Summary.

A case of effort angina of two years' duration probably associated with hypertension. Infarction occurred and the patient died in about 36 hours. The electrocardiogram showed right bundle branch block and an anterior infarct.

Figure 69. Case No. 8. P.W. Sections 3 and 6 of Figure 68 enlarged.

The haemorrhagic patches in the infarct indicate that it is a recent one, but probably older than the 36 hours clinical estimation, judging by Mallory et al. (1939). It may be that certain of the resting anginal attacks preceding the main one indicated the onset of infarction.

Comment.

There is good correlation between the electrocardiographic and pathological evidence.

The electrocardiogram corresponds almost exactly to one shown by Wood (1950) to illustrate anterior infarction complicated by right bundle branch block.

Case No. 9.

K.I., a schoolmaster aged 52, was admitted to Hospital 7th February 1951, complaining of chest pain and breathlessness for 5 days.

His father had died of a "stroke" at the age of 65, but the family history was otherwise negative. The patient himself had had an appendicectomy at the age of 27, and eight years previously a thyroidectomy, for thyrotoxicosis, from which he made a very good recovery.

During the morning of 2nd February, he had felt a sensation of a lump in the chest which increased in intensity. He became breathless and felt weak. After bearing this for three hours, he was sent home. The pain continued and was described as gnawing, and he became restless.

On admission, five days later, he was seen to be a well built man, slightly cyanosed and perspiring slightly. The pulse was regular (108 per minute), blood pressure 95/70. The heart was not enlarged clinically but there was a duplicated first sound.

There were no murmurs to be heard. The liver was thought to be slightly enlarged, but there was no other evidence of congestive failure. The urine showed albumin (trace only). The Kahn reaction was negative; the blood urea was 34 mgms.%.

An electrocardiogram was taken on the day of admission (Figure 70), and showed normal rhythm, with ST elevation in lead I and depression in lead III suggestive of anterior infarction.

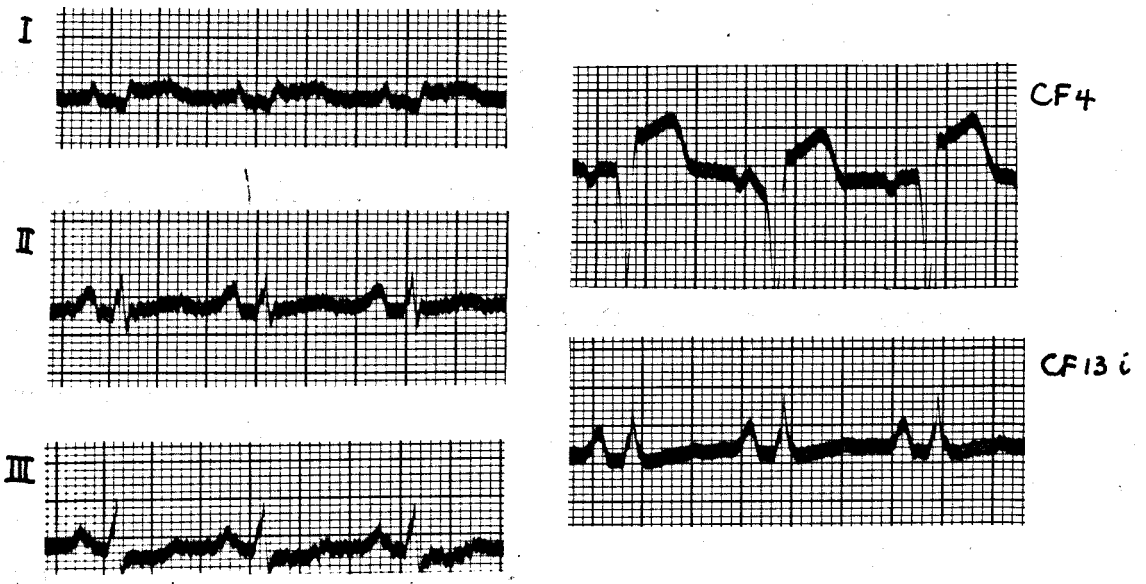


Figure 70. Case No. 9. K.I., electrocardiogram 7.2.1951.

CF4 showed a very deep Q with marked ST elevation and CF13i showed a practically normal record. The T wave was rather small but nevertheless upright.

The patient's condition worsened and he died four days after admission, nine days after the onset of infarction.

Autopsy.

There were large amounts of straw coloured fluid in both pleural cavities; none in the abdomen. The lungs showed some oedema but no consolidation.

The thyroid, with most of the lateral lobes missing was encased in fibrous tissue, and the appendix had been removed. Apart from the heart, the remaining organs appeared to be healthy. There was minimal atheroma in the abdominal aorta.

The heart weighed 570 grams. Externally, there was little to be seen apart from some roughening of the anterior surface towards the apex. This is shown in Figure 71. The anterior descending branch of the left coronary artery was blocked by thrombus about 2.5 cm. from its origin.

In this case the coronary arteries were injected with a mass consisting of two parts of a barium sulphate suspension with one of a 2% solution of sodium alginate. The barium sulphate suspension was a

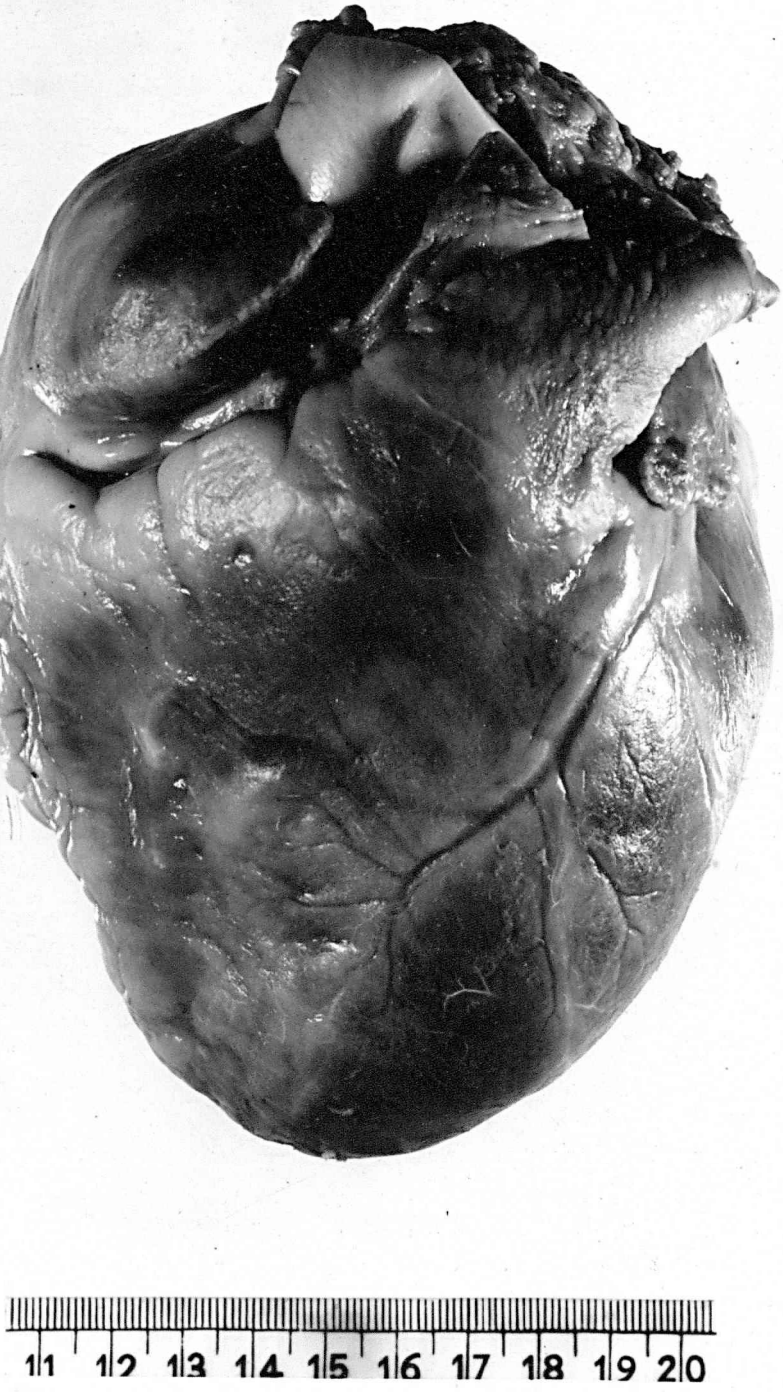


Figure 71. Case No. 9. K.I. Anterior aspect of heart.

commercial product containing one ounce by weight of the salt in one fluid ounce of suspension, and reputed to be "a dispersion of barium sulphate in a state of micro-subdivision...". It does not sediment like ordinary suspensions, and makes a convenient injection mass. The injection pressure was 180 mms. of mercury. The heart was then fixed in formol saline with added calcium chloride. The heart so prepared was radiographed, and is shown in the next Figure (72). Viewed stereoscopically no definite infarct could be located. The right ventricle showed a poor arterial supply but, unfortunately, bubbles may be seen in the right main artery. Schlesinger (1938) comments that "even in normal hearts there is a constant absence of large vessels over a small area in the posterior wall of the right ventricle near its base ...".

The heart was then cut into slices, and these were photographed and radiographed. It will be noted that the radiographs show the site of the infarct much less satisfactorily than do the photographs (Figures 73 and 75).

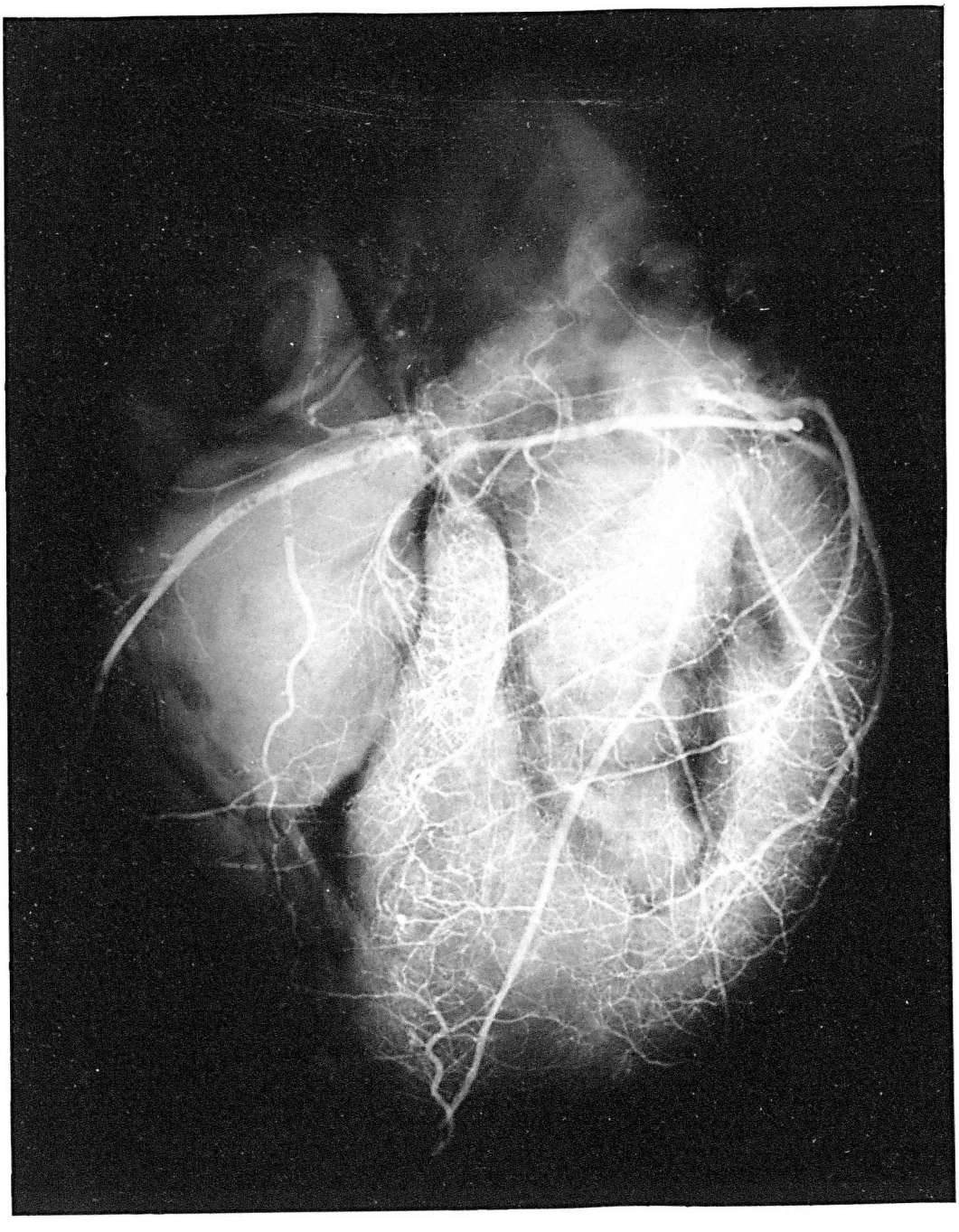


Figure 72. Case No. 9. K.I. The injected heart.

Figure 73. Case No. 9. K.I. Serial slices of the heart.

Section 1 shows a somewhat gelatinous looking area in the region of the angle between septum and anterior wall. Section 2 shows extensive infarction anteriorly and in the septum, and in Section 3 ante-mortem thrombus is adhering to the endocardium beneath. Sections 4 and 6 are enlarged in Figure 74. Section 4 shows an infarct half encircling the left ventricle and in Section 6 only part of the posterior wall appears to be unaffected. In Section 7 the ventricular cavity is completely encircled by infarct.

Summary.

A first infarction which proved fatal. The electrocardiographic evidence pointed to an anterior infarct without posterior or lateral involvement. The pathological evidence showed this to be substantially correct. The age of the infarct estimated macroscopically was compatible with the clinical data.

Comment.

There is satisfactory electrocardiographic and pathological correlation of the position of the infarct.

The case is similar to Case No. 4, but there CF13i showed slight change, which was not present here.

Figure 74. Case No. 9. K.I. Sections 4 and 6 of Figure 73.

Figure 75. Case No. 9. K.I. Radiograph of the serial slices.

Accounts have been given of nine cases of myocardial infarction, in which the diagnosis was proven at autopsy. It has been possible to correlate the position of the infarct and the electrocardipgraphic findings, using the three standard leads, an apical lead and a right pectoral lead in each case. From them there are three conclusions which may be drawn.

1. There is good correlation between the evidence of CF13i and the autopsy findings.

   Case No. 5 is the least convincing record, but there is auricular fibrillation to interfere with the electrocardiographic record and the myocardium is plainly affected by gross ischaemic lesions. CF13i fails to show any evidence of a posterior lesion although such was found at autopsy. Its appearance suggested that it was recent and may well have occurred after the electrocardiogram had been recorded.

   Cases 7, 8 and 9 show very good correlation both in a positive sense (Case No. 7)

where CF13i shows the presence of a posterior infarct; and in a negative sense (Cases No. 8 and 9) where it shows the absence of one.

Case No. 1 shows a posterior infarct in the standard leads and CF13i. That the apical lead also shows changes may be explained by the fact the infarct was so shaped that although mostly posterior there was a cup-like involvement of the apex of the left ventricle.

Cases 3 and 6 were of some interest. In the former case there was a small recent infarct in the posterior wall of the left ventricle near the septum. That no indication was shown in CF13i suggests that that lead may be of greater value in the postero-lateral infarction.

In Case No. 6 the changes in CF13i agreed with, but were no more striking than the standard leads.

Case No. 2 illustrates the value of chest leads in cases with low voltage standard leads. The correlation is good.

Case No. 4 may be considered with Case No. 3. The correlation is good and suggests that in anterior infarction CF13i may be affected if the infarct extends sufficiently far laterally in the left ventricle. This supports the view that CF13i indicates more particularly postero-lateral than posterior infarction.

In no case was there any failure of correlation between the two sources of evidence - electrocardiographic and pathological - concerning the site of the infarct.

2. Infarcts tend to be irregularly shaped, and variable in size. Those seen in the nine cases are all extensive - as to be expected in autopsy specimens. The size of the infarct in Case No. 5 is interesting as that lesion must have existed in life for a long time for such extensive calcification to occur.

In seven of the nine cases, the shape of the infarct may be likened to a small cup, corresponding to the apex of the left ventricle, with a broad elongated tongue extending upwards. This upward extension may be disposed antero-septally (Cases 5 and 9) antero-laterally (Cases 4 and 8), or posteriorly (Cases 1, 6 and 7).

3. An apical lead, such as CF4, may show evidence of an infarct when the evidence of the standard leads and CF13i points to a posterior one (Case No. 1). The explanation offered is that a "cup and tongue" infarct, with a posterior "tongue", shows changes in the apical lead owing to the electrode being adjacent to the "cup". Thus an apical lead may show anterior, or, occasionally, part of a posterior infarct. CF13i appears to be more specific in its localisation and, as indicated above, is best for showing postero-lateral lesions.

DISCUSSION

The Scope of Multiple Precordial Leads.

Electrocardiographic investigations may be resolved broadly into two categories:

a. the elucidation of arrhythmias,

b. the state of the myocardium.

It is with the second of these, and particularly in regard to the diagnosis of myocardial infarction that this work has been concerned. It may be pointed out in passing, however, that CF13i being a right pectoral lead and taken from near the right auricle therefore, shows P waves well and is generally as good as lead CR1 in the diagnosis of arrhythmias.

The abnormalities of the myocardium recognisable by electrocardiographic means are (Wilson et al. 1944; Hill, 1950; Wolff, 1950):

a. predominant ventricular hypertrophy,

b. branch bundle block,

c. myocardial infarction.

Ventricular hypertrophy.

In the diagnosis of predominant ventricular hypertrophy, the essential data are obtained by recording leads from over the right and left ventricles and from some intermediate position. Although all six precordial lead positions may be recorded three will generally suffice and leads from positions 1, 3 and 5, or 2, 4 and 6 are recommended by the American Heart Association (1943).

So far as ventricular hypertrophy is concerned the conventional precordial leads are satisfactory.

Goldberger (1944) has investigated the use of two leads, unipolar leads from the right upper abdomen and left upper scapular regions, for the diagnosis of right and left ventricular hypertrophy respectively, and has put forward criteria for these diagnoses.

Branch bundle block.

The determination of the affected, or predominantly affected, branch bundle, is made when using precordial leads, by taking a series of leads from across the precordium and ascertaining whether delay in the inscription of the intrinsic deflection takes place to the right or left of the precordium. If to the right, the right bundle is affected, if to the

left, the left bundle is at fault.

This method of diagnosing bundle block has been of inestimable value. By its means it was possible to settle the controversy which formerly took place regarding the side of the affected branch bundle.

But the six conventional precordial leads would seem to be less satisfactory than a series taken along a line perpendicular to the surface marking of the septum - a method utilised by Wilson in experimental work on dogs (Wilson et al. 1944; Wilsom, Rosenbaum & Johnston, 1947). Furthermore one would expect two leads - one from each extreme - to be adequate. Jones & Feil (1948) have investigated changes in the standard leads in cases of bundle block with change of posture. Although considerable change might occur in the standard leads, the precordial leads remained relatively unaffected, and the precordial leads chosen included the two extremes - V1 and V7, and these two are alone used for illustration of the precordial lead records.

Position 13 is not a good one for the diagnosis of branch bundle block for it is remote from a line perpendicular to the septum. Since the inverted

lead V13i or CF13i resembles a lead taken from the postero-lateral aspect of the left ventricle (position 7), it could be used to indicate the changes to the left of the septum; but this would only create confusion.

Myocardial infarction.

Here, precordial leads have, perhaps, had their most spectacular success. The pioneer work of Wilson and his colleagues has been reviewed by Hill (1950). Briefly, characteristic changes are found when an electrode is placed over a myocardial infarct, and by exploring a number of positions considerable accuracy may be achieved in the location of an infarct. Plainly, infarction in the anterior wall of the left ventricle is the most readily recognisable. Myers, Klein, Stofer and Hiratzka have published a series of eight papers (Myers, Klein, Stofer & Hiratzka, 1947; Myers, Klein & Hiratzka, 1949a; 1949b; 1949c; 1949d; Myers, Klein & Stofer, 1949) recording the correlation between electrocardiographic and pathological localisation in 161 cases of myocardial infarction. They were satisfied with the electrocardiographic localisation of anterior infarcts.

Posterior infarction can be recognised by similar criteria, as anterior infarction, if the exploring electrode be placed over the infarct. For this an oesophageal electrode is needed (Brown, 1936; Nyboer, 1941). The precordial leads by showing ST segment depression or very tall upright T waves may suggest the presence of a posterior infarct, and various other leads have been utilised. Evans & Hunter (1943) have used CR7, Rosenbaum, Wilson & Johnston (1946) have used left axillary leads from positions one or two intercostal spaces higher than the conventional precordial leads. The unipolar left leg lead is of special value for it faces the diaphragmatic (i.e. posterior) surface of the heart (Goldberger, 1947; Myers, Klein & Hiratzka, 1949c; Hill, 1950).

In the work described here, an indirect method of recording what is, in effect, sn epicardial lead from the postero-lateral aspect of the left ventricle has been described. The electrode faces the endocardial aspect of the left ventricle in its posterior part. It lies end on to the anterior wall and is relatively unaffected by that; and it appears to be

unaffected by septal changes also. By reversing its polarity, the lead may be interpreted as if it had been taken epicardially from the postero-lateral part of the heart.

The Choice of a CF Lead.

The Joint Sub-Committee of the American Heart Association and the Cardiac Society of Great Britain and Ireland made its recommendations for the use of a standardised precordial lead in 1938. Either the right arm or the left leg was suggested as the site of the indifferent electrode. Since that date there has been much discussion as to the most useful site for the placing of the indifferent electrode.

Some workers have advanced the claims of CR leads on the grounds that P waves are shown better (Barnes, 1940). This is hardly an admissable reason for leads intended to give information regarding ventricular function, especially when auricular function can be observed by using a lead from over the right

auricle (Evans, 1944). Evans (1948) also states his preference for CR leads because T wave inversion in CF1, CF6 and CF7 is common in health, and he finds that unipolar precordial leads are no better than CR leads. His views are supported by Leatham (1950).

Geiger (1939) investigated the relative value of leads IVR and IVF in 400 records and concluded that they were of equal value in 84 per cent. of cases. In the remainder IVF was superior to IVR in 13.7 per cent. and IVR superior to IVF in only 2.3 per cent. Liebow & Cushing (1941) failed to confirm these figures and concluded that in posterior infarction IVR was rather better than IVF. The reverse applied in anterior infarction.

Bain & Redfern (1948) have made a detailed study in 300 cases of the value of unipolar chest and limb leads. They confirm Geiger's view stating that there is no significant difference between CR and CF leads in 89 per cent. of cases. They go further and show that the distortion of CR or CF leads may be predicted by the appearances of the unipolar limb leads.

They quote Wilson et al. (1944) who showed that "the size of the deflections at the precordia is from three to five times that at an extremity. The

influence of the extremity electrode is, therefore, about one quarter that of the chest electrode." This relation is represented by Bain & Redfern by the equation:

$$CR = C - \frac{VR}{4} \quad \text{and} \quad CF = C - \frac{VF}{4}$$

where C is the unipolar chest lead taken from the same chest point as CR or CF. If Goldberger's augmented unipolar leads are used, the relation is:

$$CR = C - \frac{aVR}{6} \quad \text{and} \quad CF = C - \frac{aVF}{6}$$

Applying these equations to position 13 it may be seen that:

$$CF13 = V13 - \frac{aVF}{6}$$

Since position 13 is more remote from the heart than positions 1 to 6, the denominator in the third expression ($\frac{aVF}{6}$) should perhaps be smaller indicating that the leg electrode has a more pronounced effect on the bipolar lead CF13.

Now if leads CF13 and V13 be inverted the simple relations CF13 = -CF13i and V13 = -V13i obtain, and since

$$CF13i = -CF13 = -V13 + \frac{aVF}{6}$$
$$= V13i + \frac{aVF}{6}$$

The unipolar left leg lead is a valuable one in the diagnosis of posterior infarction for it faces the diaphragmatic surface of the heart. Thus in CF13i it may be seen that to the deflections of V13i the deflections of the unipolar left leg lead are added. In choosing a lead for the purpose of showing posterior infarcts the distortion introduced into this bipolar lead is of positive value.

To the theoretical advantage of CF13i one may add that the chest position is easy to locate accurately, is easy of access; and may be used in seriously ill patients in whom an oesophageal electrode would be impossible.

Chest Position 1.

The size and direction of the T wave in leads V1 and CF1 is very variable. Sigler (1944) in an analysis of 100 normal cases found T to be upright in

38 per cent., negative in 51 per cent., and diphasic in 11 per cent. Kossmann & Johnston (1935) reporting on normal values for unipolar precordial leads in 30 subjects showed that in V1 the minimum value for T was - 4.0 mms. and the maximum 5.6 mms. Deeds & Barnes (1940) have shown similar figures for CF1. Bohning et al. (1939) used CF leads with reversed polarity and using various positions of the chest electrode concluded that the transition from upward to inverted deflections occurred in the region of the sternum. Robinson, Katz & Bohning (1936) in a paper on the appearance of the T wave in lead IV in normal children and those with rheumatic heart disease show a figure which is reproduced in Figure 76.

This figure was taken from a normal, 23 year old man. The inference is that in the region of the sternum, when using CF leads, one is near the transition zone between positive and negative T waves. Further evidence of this is seen in Figure 77, which shows CF1 with both negative and positive T waves. This record can only be satisfactorily explained by assuming that respiratory movement of the heart causes the transition zone to vary. It may therefore be concluded that one

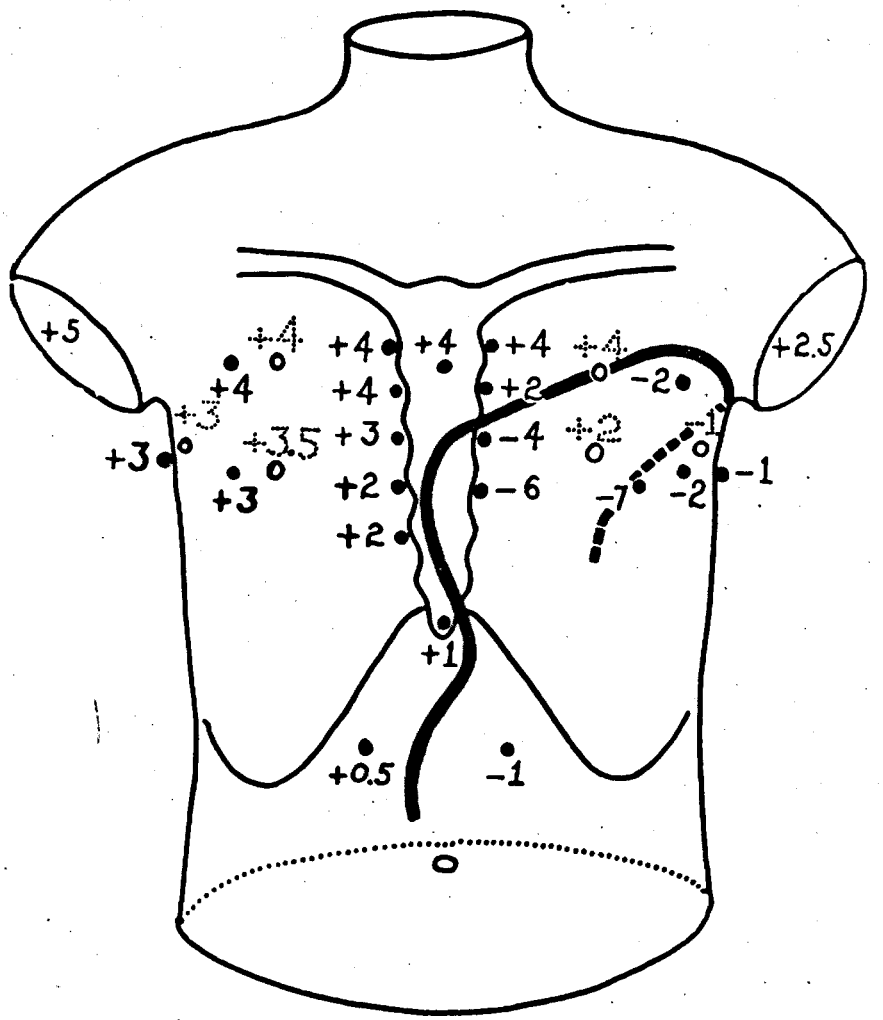


Figure 76. The line of iso-electric T waves. (Robinow, Katz & Bohning, 1936 (fig.8).

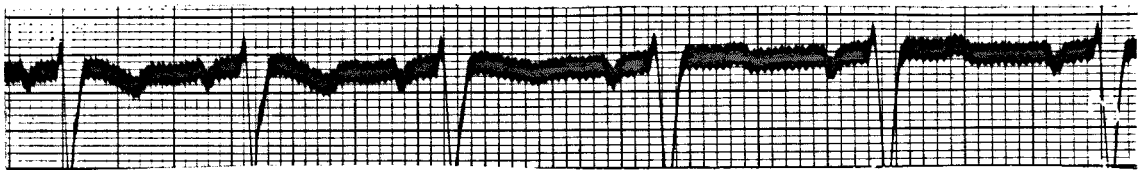


Figure 77. Variable T waves in CF1.

should take chest leads well away from the iso-electric T wave line, and CF13 being situated in the right mid-clavicular line fulfils this requirement.

Vessell & Shorr (1948) produced a paper on the right precordial lead using CF1. They state of it that "sufficient data have not yet been obtained on this lead either in regard to the range of normal or concerning its various abnormalities." They show records of two patients in whom CF1 had been obtained before and after posterior infarction; and a negative T in CF1 is shown to become positive after infarction. In 35 cases of posterior infarction T in CF1 is upright. They believe therefore that this lead is of use in the electrocardiographic diagnosis of posterior infarction. They admit the possibility that the indifferent electrode being on the left leg may facilitate these findings.

In the light of the foregoing comments on the iso-electric T wave line, it would seem advisable to

take a lead further to the right than position 1 and so obtain a constant normal T wave pattern.

The Shape of the Infarct.

Infarcts rarely conform very closely to such descriptive terms as "anterior" or "posterior". In this connexion, reference must be made to the subject of the muscle bundle localisation of infarcts.

The muscle tissue of the heart is made up of several distinct bands - the superficial and deep sino-spiral and bulbo-spiral muscles, the scroll muscle and the longitudinal muscle of the right ventricle (MacCallum, 1900; Mall, 1911; Flett, 1927). Robb & Robb (1935) have claimed that injury to one muscle band produces characteristic electrocardiographic changes, no matter what part of the muscle is damaged. The autopsy evidence advanced (Robb & Robb, 1939b) is confused, as not all cases were of myocardial infarction, but included congenital and rheumatic lesions. Also, the three standard leads have alone been considered. There is now general agreement on the inadequacy of these leads for the precise localisation of infarcts.

Lowe (1939) has drawn attention to the fact that some infarcts lie wholly within a single muscle band. This may be seen in some of the cases described above. Thus, the posterior infarct with extension to the right ventricle (Case No. 1) lies in the deep sino-spiral muscle. The old infarct in Case No. 5 and the new in Case No. 9 are both situated in the deep portion of the superficial sino-spiral muscle.

Robb & Robb (1939a) have described the blood supply to individual muscle bands. But the arterial distribution to the myocardium is variable (Barnes, 1940) and, after coronary artery occlusion, anastomoses form. ("Anastomoses always develop readily whenever and wherever arteriosclerotic narrowing or occlusion causes obstruction in the coronary artery circulation". Schlesinger, 1938). In a heart subject to ischaemic disease, one may expect the blood supply to the individual muscle bands to be irregular, and occlusions of quite small branches may affect more than one. Such hearts arriving at autopsy have usually been subjected to more than one infarction. It may be appreciated therefore that identification of the affected muscle bands is difficult. Myers et al. (1947) made no attempt to do so in their series of cases.

The Robbs' theory requires that a muscle which may be distributed anteriorly and posteriorly, in right ventricle and left, (e.g. the deep sino-spiral muscle) will produce the same electrocardiographic record, no matter in what part it is injured. This theory is at variance with the facts demonstrated experimentally by Wilson et al. (1944), using direct and semi-direct leads in dogs' hearts.

Although the identification of the injured muscle band electrocardiographically cannot be admitted, one must accept infarction in separate muscle bands as a convincing explanation for the shape assumed by infarcts. Sheldon & Sayen (1949) commenting on the work of the Robbs and Lowe, refer to the tendency of myocardial lesions to run circumferentially in the wall of the left ventricle, often assuming the shape of a thin plate. Reference to Lowe's paper (1939) shows that infarcts so shaped would occur particularly in lesions of the deep portions of the superficial sino-spiral and bulbo-spiral muscles.

Interlacing of fibres from different muscle bands occurs in the septum and especially (between superficial sino-spiral and bulbo-spiral fibres) at the apex. An explanation is thus afforded for the occurrence of

infarction at the apex irrespective of where the main part of the infarct may lie.

The Site of the Infarct.

The irregular shape of a myocardial infarct makes its position difficult to describe, for terms like "anterior" and "posterior" are approximations. They may be regarded as indicating the direction of the electrical effect of the infarct on the electrocardiogram. Terms like T_1 and T_3 introduced by Parkinson & Bedford (1928a) seem less open to objection, but they are not applicable to precordial leads. They do, however, suggest an alternative method of describing the site of an infarct, by utilising the direction of the infarct's electrical effect.

Parkinson & Bedford showed that after ST segment displacements had subsided, the residual changes in the standard leads consisted of either:

i. a negative T_1 with upright T_3, or

ii. an upright T_1 with negative T_2 and T_3.

These then were described as T_1 and T_3 infarcts; and confirmed by Bohning & Katz (1935).

These changes in the T wave are capable of being measured, and represented as vector quantities on an Einthoven triangle. They therefore possess magnitude and direction.

The magnitude may be a measure of the size of the infarct. This suggestion has not been followed up, and it is not clear how to do so, for Blumgart, Hoff, Lansdowne & Schlesinger (1937) have shown that histologically normal muscle may show electrocardiographic abnormalities.

The direction, however, of the T wave axis is an indication of the site occupied by the infarct. It will be seen in Figure 78 that an "anterior" infarct (negative T_1; positive T_3) shows rightward deviation of the T wave axis; and a "posterior" shows leftward deviation. Normal values for the T wave axis have been given by Sigler (1938) in an investigation of negative T_2 in normal hearts.

This method is not a suitable one for clinical use, where one wants an anatomical localisation, but it serves a purpose, to be indicated below. It also shows that the exact position of an infarct may be infinitely variable; and it emphasises that the electrical effect of the infarct varies with its position. "The instrument

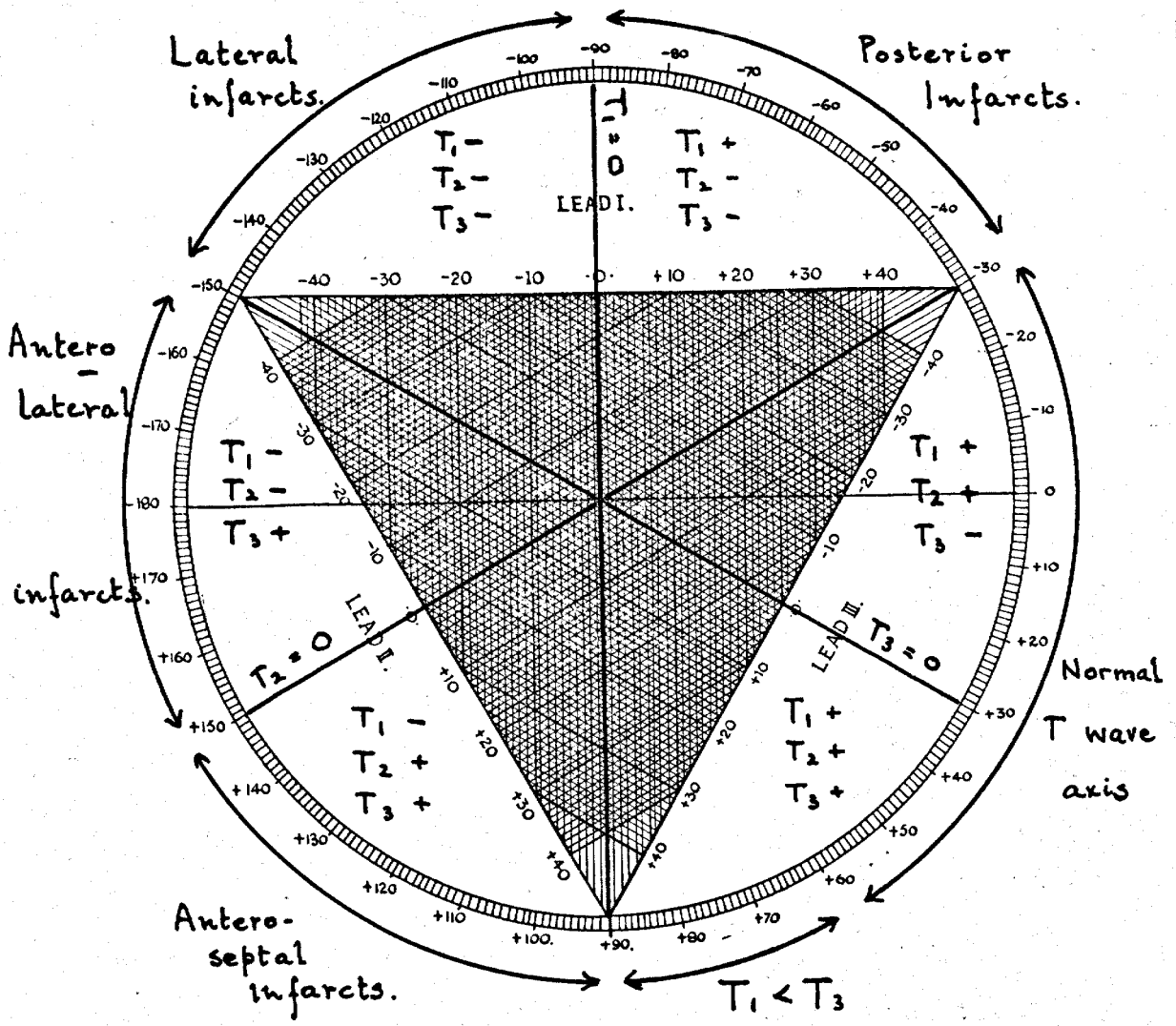


Figure 78. The association between the electrical axis of the T wave, and the site of the infarct. (Adapted from H.E.B.Pardee, "Clinical Aspects of the Electrocardiogram", 3rd ed. New York, 1933 - fig. 13).

(i.e. the electrocardiograph) essentially records physiological events. It depicts anatomical changes by inference only". (Roth, 1935).

The application of this idea to variously placed infarcts will now be considered.

Antero-septal infarcts.

These might also be described as strictly anterior infarcts.

There is a widespread notion that a standard lead electrocardiogram is normal if the T waves are all upright (QRS complexes and rhythm permitting).

Dressler (1943) and Dressler & Roesler (1948, 1949) have shown that where T_1 is smaller than T_3, although both upright, the record may be compatible with a strictly anterior infarct. This has been shown by the precordial leads.

The same relation between T_1 and T_3 may occur where there are small amplitudes of the QRS complex in lead I, as in emphysema; and in normal horizontal hearts with counter clockwise rotation around the long axis (Goldberger, 1947).

Figure 78 shows that when T_1 is smaller than T_3, the T wave axis lies between 60° and 90°, and this

zone must be regarded as a border between normal and abnormal. If T_1 is flat and T_3 is upright, the T axis is exactly 90° and this is markedly abnormal. After infarction it implies a strictly anterior infarct.

Antero-lateral infarcts of the left ventricle.

At a T wave axis of 150°, T_2 must be flat, and beyond that figure T_2 must be inverted.

So far the use of CF13i has not been mentioned in this method of localising infarcts. We have seen, however, that the T waves in CF13i and lead II follow each other fairly closely, and that when T in CF13i shows inversion as well as in the apical lead, an antero-lateral infarct is present (Case No. 4. See also Figure 8). A T wave axis lying between + 150° and - 150° signifies that T_1 and T_2 are inverted, T_3 is upright, and an antero-lateral infarct is denoted when such T waves occur after infarction.

Posterior and postero-lateral infarcts.

These may usually be diagnosed from the standard lead electrocardiogram in that T_2 and T_3 are inverted and associated with a deep Q_3 and a prominent Q_2. T_1 is upright.

Figure 78 shows that the T wave axis lies between - 30° and - 90°. At - 30° T_2 is flat; and here is the second zone between normal and abnormal T wave axes - the first being between + 60° and + 90° where the T wave axis may be normal or indicative of an antero-septal infarct. It is in such cases that CF13i is a help and although the infarct is a simple posterior one it still affects CF13i. This latter, however, shows best the presence of a postero-lateral infarct as may be seen in the autopsy cases . It has been pointed out that reversing its polarity makes it similar to a lead from the chest position 7 but superior since the latter is a semi-distant lead. Aitkenhead (1949) reports on the usefulness of V7 in postero-lateral infarcts "where it may be the only lead which overlies the infarct directly (albeit only its edge)".

Lateral infarcts of the left ventricle.

The criteria for the diagnosis of such infarcts are by no means settled, and indeed Schaffer (1944) says that there are no characteristic electrocardiographic patterns associated with this type of infarction analogous to the anterior and posterior types.

Wood, Wolferth & Bellet (1938) have given the following as signs of a lateral infarct:

a. depression of the ST interval in lead IV;

b. commonly, though not universally, depression of the ST interval in leads I and II;

c. absence of the signs of posterior infarction in lead III.

Pardee & Goldenberg (1946) in a series of 12 cases with autopsy correlation found two in which there was lateral infarction showing the above criteria. Like Wood, Wolferth & Bellet they used only one chest lead, the apical. Thomson & Feil (1944), in a critical review of the former paper, reported the results of 106 autopsy cases of myocardial infarction including 19 of lateral infarcts. Of these 9 were recent and only 4 showed Wood's criteria. The remaining five included three cases of postero-lateral infarction. Schaffer (1944) reported five cases of lateral infarction proved at autopsy with no constant electrocardiographic findings or any with Wood's.

Using multiple precordial leads, Rosenbaum, Wilson & Johnston (1946) found that leads taken in the axillary regions one or two intercostal spaces higher than the conventional chest points 4, 5 and 6 showed a high lateral infarct. Myers, Klein & Stofer (1949) confirm this finding.

Weinberg & Katz (1941) have reported the appearance of a negative T in the three standard leads following myocardial infarction (called the TN pattern because of the negative T waves). They advance seven possible explanations of which the sixth is that the infarct may be located elsewhere than in the areas favourable for the production of pure T_1 and T_3 patterns. Possible locations suggested are the lateral wall of the left ventricle, and the right ventricle.

The seven explanations offered by Weinberg & Katz are as follows (comments are made on each).

(1). Both anterior and posterior walls of the left ventricle may be involved either separately or by massive infarction. There is a tendency for both T_1 and T_3 changes to occur, hence the TN pattern.

If simultaneous anterior and posterior infarcts occur the effect on the electrocardiogram will be the vector sum of each. If diametrically opposed they will cancel out. If the anterior and posterior walls are involved by massive infarction, this must indicate the presence of a large lateral infarct.

(2). Acute infarction may occur in the presence of pre-existing coronary insufficiency either with or

without a previous infarct.

If before the recent infarct, there was an abnormal T wave axis, let us say an upright T_1 and negative T_2 and T_3, an anterior infarct will not just add inversion of T_1 to give a T_N record but will change T_2 and T_3 to less negative values.

(3). Pericarditis may produce inverted T waves in all three leads. Diffuse pericarditis may cause a T_N picture.

A comment is made later.

(4). An acute posterior wall infarction (T_3 type) may occur where there was previously left ventricular preponderance with ST depression in lead I and a negative T_1.

In such a case the inverted T_1 becomes positive.

(5). An acute anterior wall infarction (T_1 type) may occur where there was previously a negative T_3, either normal, associated with right ventricular preponderance or the residuum of an old posterior wall infarction.

In this event T_3 becomes upright after the recent infarct.

(6). An acute infarction may be located elsewhere than in the areas favourable for the production of pure T_1 and T_3 patterns. Possible atypical locations are the lateral surface of the left ventricle, and any portion of the right ventricle.

This explanation is acceptable and the former site is believed to be the correct one.

(7). When intraventricular block is present, the final as well as the initial deflection is usually abnormal in contour. A T_N type might then follow an acute myocardial infarction.

The presence of intraventricular (branch bundle) block complicates the electrocardiographic diagnosis. It is generally accepted that left branch bundle block obscures the picture of infarction (Wolff, 1950) but right branch bundle block does not. If branch bundle block is present, the T wave axis scheme may not apply.

If T is negative in the three standard leads, the T wave axis lies between - 90° and - 150°. The site suggested by Figure 78 for the location of an infarct producing such a T axis is the lateral aspect of the left ventricle, and further round the circle,

i.e. between - 30° and - 90° one enters the zone of posterior infarcts. Between - 30° and - 90° T_1 is positive and T_2 and T_3 are negative, confirming the posterior position of the infarct by accepted standards.

We see therefore that a consideration of the T wave axis places the T_N record in the lateral wall of the left ventricle.

Experimental work supporting this view has been provided by Nahum & Hoff (1945). They have studied the ST segment deviations resulting from myocardial damage due to coronary artery ligature in dogs. They found that in the standard leads:-

1. Damage to the right ventricle results in ST depression.

   If the damage is anterior there is a depressed ST in lead III;

   If posterior a depressed ST in lead I;

   If both anterior and posterior, depressed ST segments in both leads I and III.

2. Damage to the left ventricle results in ST elevation.

   If the damage is anterior, there is an elevated ST in lead I;

   If it is posterior, elevated ST in lead III;

And if both anterior and posterior,
elevation of ST in leads I and III.

3. Damage to the anterior surface of the heart (i.e. involving both ventricles).
   Depressed ST segment lead III;
   Elevated ST segment lead I.

4. Damage to the posterior surface of the heart (involving both ventricles).
   Depressed ST segment lead I;
   Elevated ST segment lead III.

When ST segment displacements subside, one finds that:

ST elevation results in an iso-electric ST with an inverted T;

ST depression results in an iso-electric ST with a taller upright T than previously.

Applying this to Nahum & Hoff's work one may anticipate:

1. Damage to the right ventricle produces later
   anteriorly, a tall upright T_3
   posteriorly, a tall upright T_1
   both anteriorly and posteriorly, tall upright T_1 and T_3.

2. Damage to the left ventricle produces later
   anteriorly, inverted T_1
   posteriorly, inverted T_3
   both anteriorly and posteriorly, inverted T_1 and T_3 (the T_N pattern).
3. Damage to the anterior surface of the heart (i.e. involving both ventricles) produces later
   inverted T_1
   upright T_3
4. Damage to the posterior surface of the heart produces
   upright T_1
   inverted T_3

It may be seen that 3 and 4 are in accordance with known facts; and 1 and 2 may be expected to apply also. In 2 we see that damage to the whole of the left ventricle should produce inverted T waves in all three standard leads - a state existing also in pericarditis. This will be a subject of comment later. Figure 9 is to be regarded as an example of lateral infarction.

Infarction of the right ventricle.

The work of Nahum & Hoff quoted above is cited to show that lateral infarction of the left ventricle gives rise to negative T waves in the standard leads. It may also be quoted to indicate the expected changes in experimental infarction of the right ventricle , namely ST depression in all standard leads with upright T waves as the ST segment depressions subside. Provided that T_1 is greater than T_3, the final T wave axis is thus normal.

It is worth comment that the T wave axis in the Einthoveh triangle shown in Figure 78 might be expected to show either right ventricular infarction or isolated infarction of the interventricular septum when it lies between + 60° and - 30°. This is a possible explanation for failure to recognise right ventricular infarction electrocardiographically.

Korey & Katz (1934) had carried out similar work to Nahum & Hoff, but had produced myocardial damage by injecting alcohol in various situations of dogs' hearts. As a result they described four types of infarct - the T_1, T_3, T_N and T_P - the last named having positive T waves in the standard leads preceded

by ST depression and therefore comparable to records obtained by Nahum & Hoff from the right ventricle. They concluded that the electrocardiographic changes do not depend upon the location of the injured area, and they went further by saying that using the standard three leads the electrocardiogram does not differentiate between:

1. injury to the anterior and posterior walls of the ventricles;
2. injury to the right and the left ventricles;
3. injury to apical and basal portions of the ventricles.

It is generally agreed now that a distinction can be made between anterior and posterior infarcts, and Katz (1946) has since abandoned this view quoted above. It may therefore be concluded that this experimental work is untenable.

But even if one could be certain of the electrocardiographic changes of right ventricular damage from experimentation, it still remains to show that this occurs in man; and here evidence is remarkable for its paucity. Infarcts of the antero-septal and postero-septal regions of the left ventricle are sometimes

continued into the adjacent parts of the right ventricle. The frequency with which this occurs varies considerably in different reports. Applebaum & Nicolson (1935) found eight cases of involvement of the right ventricle out of 118 cases in which infarcts were localised. Each of the eight cases showed involvement of the left ventricle also. Bean (1938) and Feil, Cushing & Hardesty (1938) believe that involvement of the right ventricle (as an extension from the left) may occur in up to one-third of cases examined. Barnes & Ball (1932) regard extension into the right ventricle as uncommon. There is no convincing record, however, of isolated infarction of the right ventricle other than as an extension. Myers, Klein & Hiratzka (1949b) examined 161 cases at autopsy and were unable to find one. Bosco (1947) states (dogmatically) that infarction of the right ventricle does not occur.

There is supporting evidence for this view of Bosco's. Schlesinger (1938) has already been quoted as stating that in normal hearts there is a constant absence of large vessels over a small area in the posterior wall of the right ventricle near the base. He states that this apparently avascular area is thin and never fibrosed and suggests that it may be nourished

by Thebesian vessels. Lendrum, Kondo & Katz (1945) have shown that in dogs "Thebesian drainage into the right ventricle is an important element in coronary drainage. Furthermore, it supports our idea --- that these Thebesians in the right ventricle may serve as immediate portals of entry of blood when the coronary arteries feeding the right ventricle are suddenly or gradually occluded."

The electrocardiographic evidence of right ventricular infarction in man therefore must be regarded as undetermined.

It may now be queried as to what connection there is between lead CF13i and localisation of infarcts using the T wave axis. It is this: that CF13i bears a relation to standard lead II; and as a result a distinction between antero-septal and antero-lateral infarcts may be made in the standard leads by the direction of T_2. Hence the T wave axis of the standard leads differentiates between antero-septal and antero-lateral infarcts.

Factors modifying the T wave axis localisation of infarcts.

1. Pericarditis.

The electrocardiographic changes of pericarditis are due to the myocardial lesion causing it (Peel, 1934). When this occurs in myocardial infarction it may be localised to the epicardial aspect of the infarct or it may be generalised. In the former case it has no effect on the electrocardiogram (Wood, 1950). In the latter it may do so by producing

a. pericardial effusion with production of low voltage (Scherf & Boyd, 1948), or

b. a generalised irritation of the whole of the surface of the heart. The currents of injury in such a case, when added as vector quantities, give a resultant directed away from the left lateral surface of the heart (Burch & Winsor, 1945). As shown in Figure 78 this results in T wave inversion in all standard leads and this is in accordance with known facts. The electrocardiographic picture

of generalised pericarditis is one of ST elevation in all standard leads followed by T inversion.

It follows that if generalised pericarditis should follow an infarct it would affect the standard lead electrocardiogram in such a way as to make the infarct appear more lateral than in effect is really would be.

2. Posture.

Just as alteration in the posture of the patient may affect the QRS axis, so may it affect the T axis (Sigler, 1938). Hence, alterations in heart position due to posture, or phase of respiration, might affect the localisation of an infarct.

This is not a serious objection for the effect is relatively small. Furthermore electrocardiograms are recorded with the patient either sitting or lying down so that postural changes do not occur. The T wave axis is not in clinical use, but has been used above as a means of indicating how the standard lead may be anticipated.

Summary and Conclusions

A description has been given of a series of chest electrocardiograms and of how one appeared to be useful in the diagnosis of myocardial infarction.

Nine cases have been described in which this lead, a right pectoral lead, had been recorded before death and the changes shown have been correlated with the autopsy findings. This lead is of especial value in the diagnosis of postero-lateral infarcts.

As a result of the examination of the heart in the nine cases, certain comments are made on the shape assumed by the myocardial infarcts. Their relation to the component muscles of the heart is discussed.

As a result of the electrocardiographic localisation of infarcts, using the apical and right pectoral leads, a method is suggested whereby the three standard leads may be used in the more precise localisation of infarcts. The electrocardiographic

diagnosis of infarction of the lateral wall of the left ventricle is discussed, and also the subject of right ventricular infarction.

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REFERENCES

Aitkenhead, A.C. (1949). Brit. Heart J., 11, 94.

American Heart Association (1938). Amer. Heart J., 15, 235.

--- (1943). Amer. Heart J., 25, 535.

--- & Cardiac Society of Great Britain & Ireland (1938). Amer. Heart J., 15, 107; Brit. Heart J., 1, 1939, 45.

Applebaum, E., Nicolson, G.H.B. (1935). Amer. Heart J., 10, 662.

Bain, C.W.C., Redfern, E.McV. (1948). Brit. Heart J., 10, 9.

Barnes, A.R. (1940). "Electrocardiographic Patterns", Thomas, Springfield.

--- , Ball, R.G. (1932). Amer. J. med. Sci., 183, 215.

--- , Whitten, M.B. (1929). Amer. Heart J., 5, 142.

Bean, W.B. (1938). Ann. int. Med., 12, 71.

Blumgart, H.L., Hoff, H.E., Lansdowne, M., Schlesinger, M.J. (1937). Amer. J. med. Sci., 194, 493.

Bohning, A., Katz, L.N. (1935). Amer. J. med. Sci., 189, 833.

--- --- , Robinow, M., Gertz, G. (1939). Amer. Heart J., 18, 25.

--- --- , Langendorf, R. (1941). Amer. Heart J., 22, 778.

Bosco, G. (1947). Amer. Heart J., 33, 722.

Brown, W.H. (1936). Amer. Heart J., 12, 1; 307.

Carter, E.P., Richter, C.P., Greene, C.H. (1919). Johns Hopk. Hosp. Bull., 30, 162.

Crawford, J.H., Roberts, G.H., Abramson, D.I., Caldwell, J.C. (1932). Amer. Heart J., 7, 627.

Deeds, D., Barnes, A.R. (1940). Amer. Heart J., 20, 261.

Dressler, W. (1943). Amer. Heart J., 26, 313.

--- , Roesler, H. (1948). Amer. Heart J., 35, 58.

--- --- (1949). "Atlas of Electrocardiography", Oxford.

Edwards, J.C., Van der Veer, J.B. (1938). Amer. Heart J., 16, 431.

Einthoven, W. (1903). Ann. Phys., Lpz., Folge IV, 12, 1059.

Eppinger, H., Rothberger, C.J. (1909). Wien. klin. Wschr., 22, 1091.

Evans, W. (1944). Brit. Heart J., 6, 221.

--- , Hunter, A. (1943). Brit. Heart J., 5, 73.

Feil, H., Cushing, E.H., Hardesty, J.T. (1938). Amer. Heart J., 15, 721.

Flett, R.L. (1927). J. Anat., 62, 439.

Geiger, A.J. (1939). Amer. Heart J., 18, 715.

Goldberger, E. (1944). Amer. Heart J., 28, 621.

--- (1947). "Unipolar Lead Electrocardiography", London.

Hamman, L. (1926). Bull. Johns Hopk. Hosp., 38, 273.

Herrick, J.B. (1912). J. Amer. med. Ass., 59, 2015.

--- (1919). J. Amer. med. Ass., 72, 387.

Hill, I.G.W. (1950). Lancet, 1, 985; 1027.

Hirschboeck, F.J. (1934). Amer. Heart J., 10, 264.

Hoffmann, A.M., Delong, E. (1933). Arch. int. Med., 51, 947.

Johnston, F.D., Hill, I.G.W., Wilson, F.N. (1935). Amer. Heart J., 10, 889.

Jones, A.M., Feil, H. (1948). Amer. Heart J., 36, 739.

Katz, L.N. (1946). "Electrocardiography", 2nd ed., Philadelphia.

--- , Kissin, M. (1933). Amer. Heart J., 8, 595.

Korey, H., Katz, L.N. (1934). Amer. J. med. Sci., 188, 387.

Kossmann, C.E., de la Chapelle, C. (1938). Amer. Heart J., 15, 700.

--- --- (1939a). Amer. Heart J., 18, 344.

--- --- (1939b). Amer. Heart J., 18, 352.

--- , Johnston, F.D. (1935). Amer. Heart J., 10, 925.

Leatham, A. (1950). Brit. Heart J., 12, 213.

Lendrum, B., Kondo, B., Katz, L.N. (1945). Amer. J. Physiol., 143, 243.

Levine, S.A. (1929). Medicine, 8, 245.

Liberson, A., Liberson, F. (1933). Ann. int. Med., 6, 1315.

Liebow, I.M., Cushing, E.H. (1941). Amer. Heart J., 22, 125.

Lowe, T.E. (1939). J. Path. Bact., 49, 195.

MacCallum, J.B. (1900). Johns Hopk. Hosp. Rep., 9, 307.

MacNee, J.W. (1925). Quart. J. Med., 19, 44.

Mall, F.P. (1911). Amer. J. Anat., 11, 211.

Mallory, G.K., White, P.D., Salcedo-Salgar, J. (1939). Amer. Heart J., 18, 647.

Marchant, E.W., Wallace-Jones, H. (1940). Brit. Heart J., 2, 97.

Myers, G.B., Klein, H.A., Hiratzka, T. (1948). Amer. Heart J., 36, 838.

--- --- --- (1949a). Amer. Heart J., 37, 205.

--- --- --- (1949b). Amer. Heart J., 37, 720.

--- --- --- (1949c). Amer. Heart J., 38, 547.

--- --- --- (1949d). Amer. Heart J., 38, 837.

--- --- , Stofer, B.E. (1948). Amer. Heart J., 36, 535.

--- --- --- (1949). Amer. Heart J., 37, 374.

--- --- --- , Hiratzka, T. (1947). Amer. Heart J., 34, 785.

Nahum, L.H., Hoff, H.E. (1945). Amer. J. Physiol., 143, 723.

Nyboer, J. (1941). Amer. Heart J., 22, 469.

Pardee, H.E.B. (1920). Arch. int. Med., 26, 244.

--- , Goldenberg, M. (1946). Arch. Inst. Cardiol. Mexico, 16, 109.

Parkinson, J., Bedford, D.E. (1928a). Heart, 14, 195.

--- --- (1928b). Lancet, 1, 4.

Peel, A.A.F. (1934). Glasgow med. J., 122, 149.

Robb, J.S., Robb, R.C. (1935). Amer. Heart J., 10, 287.

--- --- (1939a). Amer. J. med. Sci., 197, 7.

--- --- (1939b). Amer. J. med. Sci., 197, 18.

Robinson, R.W., Contratto, A.W., Levine, S.A. (1939). Arch. int. Med., 63, 711; 732.

Rosenbaum, F.F., Hecht, H., Wilson, F.N., Johnston, F.D. (1945). Amer. Heart J., 29, 281.

---, Wilson, F.N., Johnston, F.D. (1946). Amer. Heart J., 32, 135.

Roth, I.R. (1935). Amer. Heart J., 10, 798.

Saphir, O., Priest, W.S., Hamburger, W.W., Katz, L.N. (1935). Amer. Heart J., 10, 567; 762.

Sayen, J.J., Sheldon, W.F. (1949). Amer. Heart J., 38, 688.

Schaffer, C.F. (1944). Amer. Heart J., 28, 39.

Scherf, D., Boyd, L.J. (1948). "Clinical Electrocardiography", 3rd ed., London.

Schlesinger, M.J. (1938). Amer. Heart J., 15, 528.

Sheldon, W.F., Sayen, J.J. (1949). Amer. Heart J., 38, 517.

Sigler, L.H. (1938). Amer. Heart J., 15, 146.

--- (1944). "The Electrocardiogram", London.

Smith, F.J., Goodrich, B.E., Needles, R.J. (1939). J. Lab. clin. Med., 24, 367.

Smith, F.M. (1918). Arch. int. Med., 22, 8.

--- (1920). Arch. int. Med., 25, 673.

--- (1923). Arch. int. Med., 32, 497.

Sprague, H.B., Orgain, E.S. (1935). New Engl. J. Med., 212, 903.

Thomson, H.W., Feil, H. (1944). Amer. J. med. Sci., 207, 588.

Vesell, H., Shorr, B. (1948). Brit. Heart J., 10, 158.

Weinberg, H.B., Katz, L.N. (1941). Amer. Heart J., 21, 699.

Whitten, M.B. (1928). Arch. int. Med., 42, 846.

--- (1930). Arch. int. Med., 45, 383.

Wilson, F.N. (1930). Amer. Heart J., 5, 599.

---, Hill, I.G.W., Johnston, F.D. (1934). Amer. Heart J., 9, 596.

--- --- --- (1935). Amer. Heart J., 10, 903.

---, Johnston, F.D., Hill, I.G.W. (1935). Amer. Heart J., 10, 1025.

--- ---, MacLeod, A.G., Barker, P.S. (1934). Amer. Heart J., 9, 477.

--- ---, Rosenbaum, F.F., Erlanger, H., Kossmann, C.E., Hecht, H., Cotrim, N., de Oliviera, R., Barker, P.S. (1944). Amer. Heart J., 27, 19.

---, MacLeod, A.G., Barker, P.S. (1932). Amer. Heart J., 7, 305.

---, Rosenbaum, F.F., Johnston, F.D. (1947). "Interpretation of the Ventricular Complex of the Electrocardiogram" In "Advances in Internal Medicine", 2. New York.

Wolferth, C.C., Wood, F.C. (1932). Amer. J. med. Sci., 183, 30.

Wolff, L. (1950). "Electrocardiography; fundamentals and clinical application". Philadelphia & London.

Wood, F.C., Wolferth, C.C. (1933). Arch. int. Med., 51, 771.

--- --- , Bellet, S. (1938). Amer. Heart J., 16, 387.

Wood, P. (1950). "Diseases of the Heart and Circulation", London.

--- , Selzer, A. (1939). Brit. Heart J., 1, 49.
